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HEART DISEASE AND THORACIC ANEURYSM

F. J. POYNTON

NEW YORK: THE C. V. MOSEBY COMPANY, 1910

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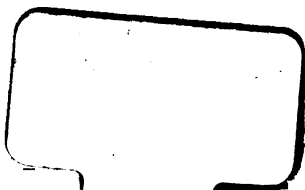
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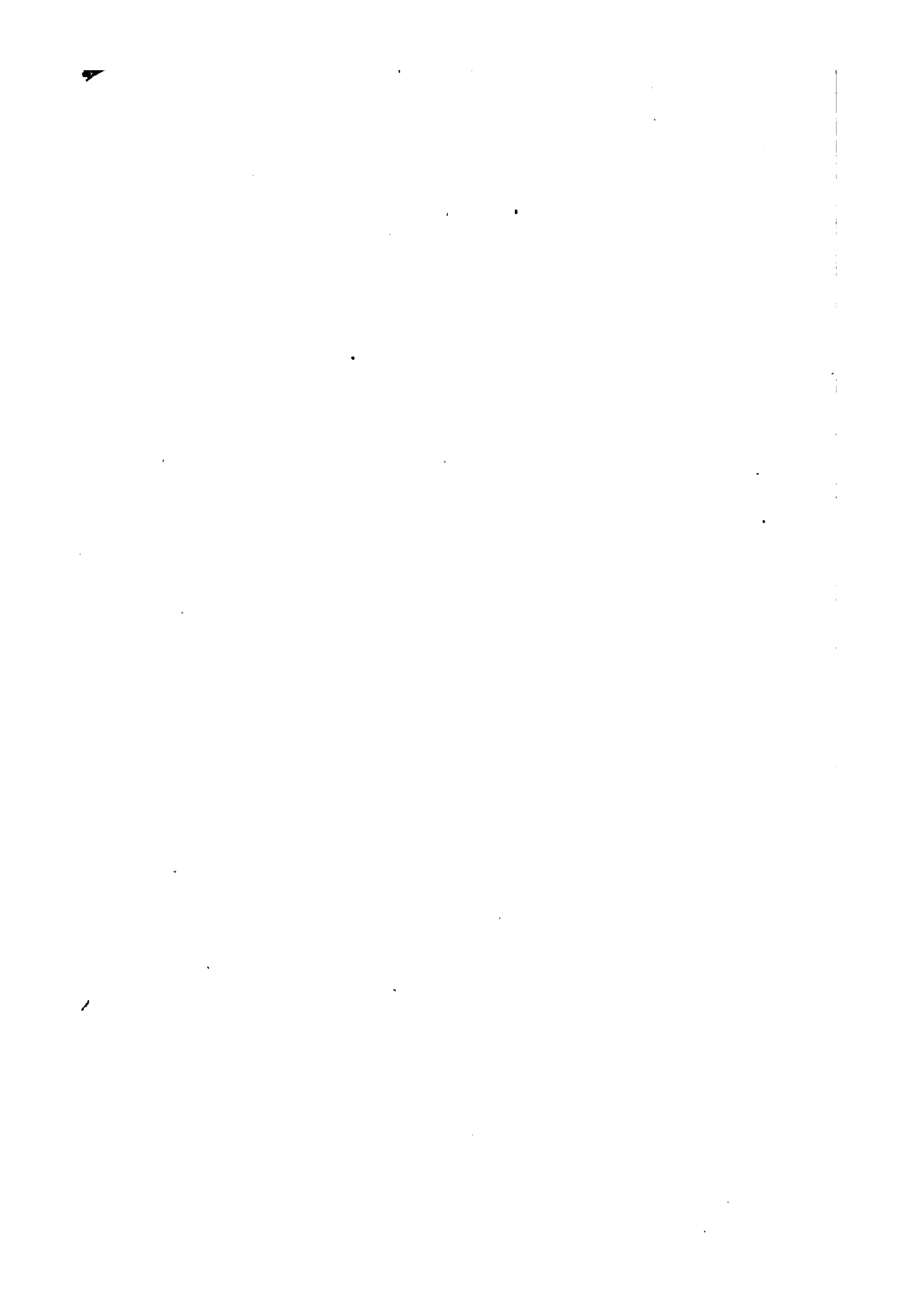
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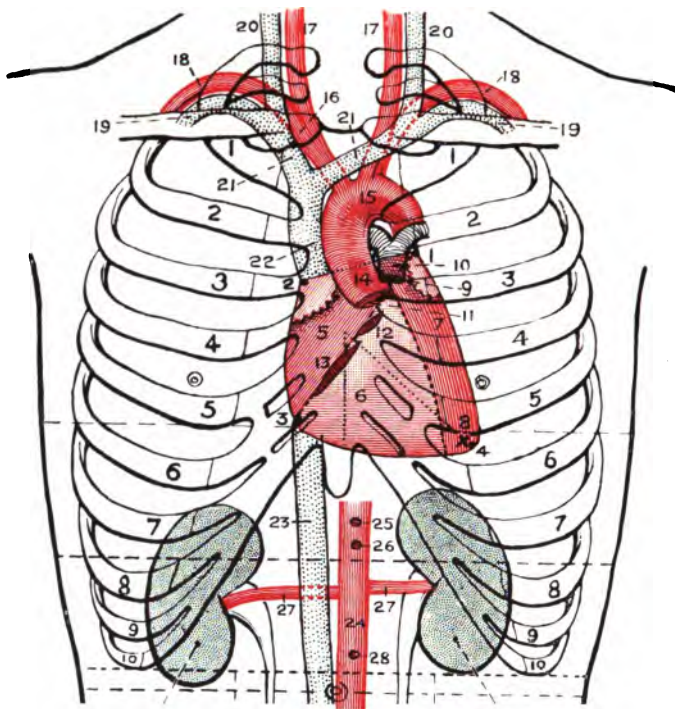


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The Topography of the Heart and Great Vessels.

From Rawling, 'Landmarks and Surface Markings,' by permission of H. K. Lewis.

- | | |
|---|--|
| 1-4. The four points of the heart. | 19. The right and left subclavian veins. |
| 5. The auricular area. | 20. The right and left internal jugular veins. |
| 6. The ventricular area. | 21. The right and left innominate veins. |
| 7. The left ventricle. | 22. The superior vena cava. |
| 8. The apex beat. | 23. The inferior vena cava. |
| 9. The pulmonary valve. | 24. The abdominal aorta. |
| 10. The pulmonary artery. | 25. The coeliac axis. |
| 11. The aortic valve. | 26. The superior mesenteric artery. |
| 12. The mitral valve. | 27. The renal arteries. |
| 13. The tricuspid valve. | 28. The inferior mesenteric artery. |
| 14. The ascending aorta. | 29. The common iliac arteries. |
| 15. The aortic arch. | 30. The internal iliac arteries. |
| 16. The innominate artery. | 31. The external iliac arteries. |
| 17. The right and left common carotid arteries. | 32. The kidney. |
| 18. The right and left subclavian arteries. | |

OXFORD MEDICAL PUBLICATIONS

HEART DISEASE AND THORACIC ANEURYSM

BY

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P R E F A C E

AN attempt has been made in this book to describe in outline the most important forms of heart disease, together with the chief methods that are employed in their clinical investigation and treatment. The usual arrangement of the subject has been somewhat departed from, and after the chapters upon Clinical Methods, a description has been given of Rheumatic Morbus Cordis, with the intention of providing one complete picture of heart disease, rather than sketches of the isolated lesions of pericarditis and endocarditis. I think that by this arrangement the subject becomes less complicated and the various cardiac affections are more easily understood.

In an Appendix a series of prescriptions has been given to which numerical reference is made in the text. This is not intended to give the impression that treatment can be learnt from books, or that rule-of-thumb prescription is to be encouraged, but to help the beginner by illustrations of definite combinations and doses of drugs.

To the writings of others I am much indebted. My thanks are due to the Editors of the *Lancet*, the Royal Medico-Chirurgical and Pathological Societies, Messrs. Lea Brothers, and J. B. Lippincott & Co. for permission

to use some of the original plates that have illustrated articles upon Rheumatic Fever published from time to time by Dr. Paine and myself. To Mr. L. Rawling I am obliged for the first plate. Mr. Lawrence, Curator of the Museum of University College, London, has most kindly made me a sketch of the heart, showing the bundle of His; and Dr. T. Lewis has provided the sphygmographic tracings. Mr. Otto May has given me valuable assistance with the proofs and with suggestions; and my best thanks are due to the Editor and publishers for the skill and care with which they have produced this book.

F. J. POYNTON.

1 HARLEY PLACE, W.

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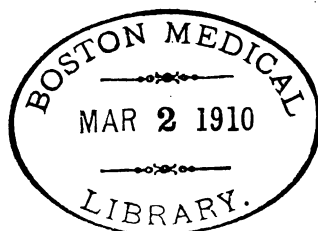
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CHAPTER I

THE HEART AND PERICARDIUM

Part I. Anatomical. Surface-markings—The pericardium—The cardiac wall—The A. V. bundle of His—The endocardium and valves—The innervation of the heart—The relations of the thoracic aorta.

Part II. Physiological. The cardiac sounds—The cardiac cycle—The cardiac nerves—Influence of the respiratory movements on the heart—The heart-beat—Heart-block.

Part III. Experimental Cardiac Pathology. Investigation of cardiac rhythm—Myocardial disease—Carditis—Arteritis.

1. SURFACE-MARKINGS

The heart and pericardium are situated in the middle mediastinum and lie nearer to the front than the back of the chest. There is, however, but little of the pericardium in contact with the anterior wall, for the pleurae and lungs intervene and cover the large vessels, and all but a small and variable surface of the right ventricle.

The obliquity of the axis of the heart is a little difficult to realize at the bedside, and there is a decided tendency to picture the heart as though it were almost vertical in position when, as a matter of fact, the long axis forms an angle of 40° with the horizontal.

These four lines will for practical purposes define the relation of the heart to the anterior chest wall.

1. *The upper line.* This extends from one inch to the right of the sternum to one inch to the left at the level of the

2 THE HEART AND PERICARDIUM

second intercostal space. It touches the right auricle and its appendix, the pulmonary conus and tip of the left auricular appendix.

2. *The right line.* This is a curved line drawn from the right limit of the preceding to the sixth chondro-sternal junction. The convexity of the curve is to the right and its summit is at the level of the fourth intercostal space, and extends two inches from the mid-sternal line.

This limit is formed by the right auricle.

3. *The left line.* This is also curved and is drawn from the left extremity of the upper line, downward to the apex beat, which is situated in the fifth intercostal space $3\frac{1}{2}$ inches from the middle line and just internal to the end of the fifth rib.

It is formed almost entirely by the left ventricle.

4. *The lower line.* This is represented by a line drawn from the sixth right chondro-sternal junction to the apex beat.

It is formed by the right ventricle.

The position of the heart varies with posture and with the respiratory movements, but of more importance is the influence of *age and growth*.

In childhood the heart is proportionately broader and is placed further to the left of the middle line. As a result, under the age of twelve years, the apex beat is usually in the fourth intercostal space. Again, in children under twelve years the impulse is often found in the vertical nipple line and not internal to it as in the adult, and under six years it frequently extends outside the vertical nipple line.

There are numerous other surface-markings in connexion with the heart, for which reference must be made to treatises upon anatomy. Here, emphasis will be laid only upon certain points which have struck me as liable to be overlooked.

The front of the heart is almost entirely represented by the *right auricle and ventricle*, a small strip only of the left ventricle appearing upon the extreme left.

The position of the pulmonary conus of the right ventricle is sometimes not thoroughly realized, and the left auricle thought to lie more in front than is actually the case. The following surface-marking is useful. A line drawn from the sixth right chondro-sternal junction, upward to the third costal cartilage on the left side, marks the furrow between the right ventricle and right auricle and thus acts as a guide to the pulmonary conus.

The base of the heart posteriorly, corresponds to the space between two lines drawn at the level of the sixth and eighth dorsal vertebrae.

2. THE ANATOMY OF THE PERICARDIUM

Such details only are considered as have close bearing upon the subject of heart disease.

The Pericardium. This sac is conical in shape with its base firmly attached to the central tendon of the diaphragm. Above, the fibrous layer is prolonged on to the large vessels and is lost in their outer coats. The pulmonary artery and aorta where they are united are entirely enveloped by the pericardium. This relation is important in the study of *aneurysm of the aorta*, for aneurysms of the intrapericardial part, enclosed as they are in the sac, cannot reach a large size.

The pericardium forms but a *loose investment*, and when there is inflammation and exudation into the cavity the roomy spaces around the heart become distended. The pericardium then bulges out into two pockets beyond the

right and left limits of the heart immediately above the diaphragm. It is also distended upwards, and to the left of the sternum, reaching in extreme cases as high as the left clavicle. Lastly, when the patient is recumbent a considerable collection of fluid may collect posteriorly.

Under the serous layer of the pericardium, and especially in the grooves of the heart, there are accumulations of *fat*, and these in the obese may be greatly increased and become a source of danger by their embarrassing effect upon the cardiac action.

In structure the parietal pericardium consists essentially of dense, unyielding, interlacing, fibrous tissue, lined on the inner aspect with endothelium. The serous layer reflected on the heart is also formed of connective tissue with elastic fibres, and is lined on its outer surface with endothelium.

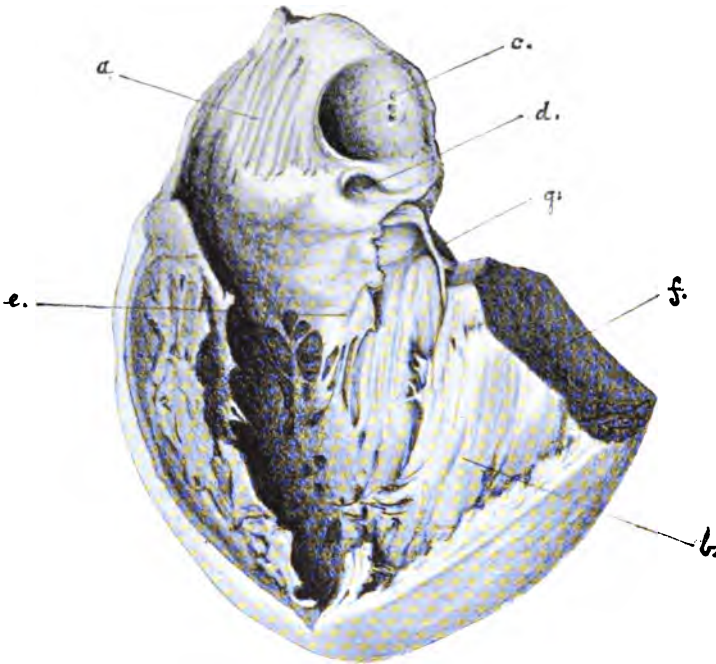
There are numerous blood capillaries in the looser *subendothelial tissues*, and it is to these positions that micrococci are chiefly carried in the various infective inflammations of the pericardium.

3. THE CARDIAC WALL

The wall of the heart is formed by masses of muscle bound together by strands of connective tissue containing blood vessels, lymphatics, and nerves. In disease, and particularly in infective diseases of the heart, the pathological changes in the functional tissue—the muscle—must be distinguished from those in the supporting tissues.

In the muscle are seen the effects produced by the special actions of the various *toxins*. In the connective tissue occur *inflammatory reactions*, which, if chronic, tend to produce a fibrosis of the cardiac wall.

PLATE I



Part of a heart, including a portion of the wall of the right auricle, (a) and the muscular septum ventriculorum, (b) seen from the right side; (c) orifice of the inferior vena cava; (d) orifice of the coronary sinus; (e) septal flap of the tricuspid valve. The anterior part of the septal flap has been removed, together with the adjacent part of the auricular endocardium above it. The infundibular part (f) of the muscular septum has been cut through at a level with the pars membranacea, and the latter structure removed, thus displaying the rounded upper margin (g) of the muscular septum and the auriculo-ventricular bundle of His immediately above it.

From an original drawing by Mr. T. W. P. Lawrence, Curator of the Museum, University College Hospital.

The myocardium is exceedingly vascular, minute blood vessels penetrating every part of the musculature. The lymphatics also are very extensively distributed beneath the endocardium and pericardium, and between the muscular fibres, and in cases of severe pericarditis it is the rule to find that the *lymphatic glands* in connexion with the heart and pericardium are considerably enlarged and inflamed.

4. THE AURICULO-VENTRICULAR BUNDLE OF HIS

In the last few years a great deal of attention has been directed to this particular part of the cardiac muscle. In the condition termed *heart-block*, associated with the Stokes-Adams Syndrome, vide p. 249, this muscular bundle is believed by many to be of particular importance.

In this brief account of its anatomy the description given by Tawara, who has investigated this structure with indefatigable labour, is followed. The bundle commences close to the anterior edge of the *coronary vein*, and passes forward below the foramen ovale on the right side of the auricular septum, and immediately above the *insertion of the median flap of the tricuspid valve*. Here it forms a knotlike thickening, which is produced by the twisted muscular fibres. From the knot a process arises, which penetrates *the fibrous part of the septum* between the two ventricles immediately below the undefended space. At this point it divides into *two main branches*, a right and left, both of which run down in the interventricular septum immediately beneath the septal endocardium. Both these branches descend vertically to the lower third of the ventricle, and then some of the fibres pass into the anterior and posterior *papillary muscles* of the respective ventricles. Others pass into the ventricular

trabeculae, and then, spreading upward and downward line the *whole inner surface* of the ventricles, fusing eventually with the other cardiac muscular fibres.

This bundle has a *peculiar structure*: the fibres interlace and fuse with one another, and are characterized by less development of the sarcoplasm, than in the ordinary cardiac muscle. In the ventricular part of the bundles the fibres are large and closely packed, and stain feebly on account of the reduced fibrillary substance. They are, in fact, identical with those mysterious fibres that Purkinje demonstrated in the heart of the sheep. Aschoff states that this bundle does not hypertrophy with the other muscles in cardiac disease.

The function that is attributed to this bundle is the conduction of the *auricular contractions to the ventricles*, these fibres forming the only direct muscular connexion between these chambers.

In the modern writings upon this structure the abbreviated term A.V. bundle is often employed.

5. THE ENDOCARDIUM AND VALVES

The endocardium lines all the cavities of the heart and the surface of the valves, and merges into the intima of arteries and veins.

It is to the tissues immediately beneath the endocardium that the terminations of the auriculo-ventricular bundle have been traced, and thus recognized as identical with Purkinje's fibres.

In the subendocardial tissue there may also be a considerable deposit of fat.

The Cardiac Valves. The anatomical and clinical surface-markings.

1. *The anatomical.* A small circle will enclose the four

surface-markings of the valvular orifices. They are from above downward indicated by these points :—

(1) *The pulmonary*, immediately to the left of the sternum at the upper border of the third costal cartilage.

(2) *The aortic*, behind the sternum at the lower border of the third costal cartilage.

(3) *The mitral*, immediately to the left of the sternum at the level of the fourth costal cartilage.

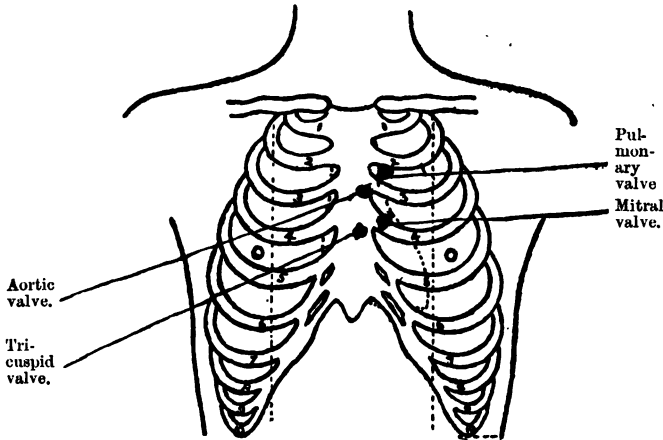


FIG. 1. A diagram to show the surface-markings of the cardiac valves.

(4) *The tricuspid* behind the sternum at the level of the fourth intercostal space.

In studying diseases of the valves, there are certain areas which are of especial importance, and which may be termed the clinical surface-markings. These will be better appreciated after reading Chapter iii. Here they will be given for comparison with anatomical surface-markings.

2. *Clinical surface-markings.*

(1) *The pulmonary area.* The inner end of the third left costal cartilage and adjoining space.

(2) *The aortic area.* The inner end of the second right intercostal cartilage and adjoining space.

(3) *The mitral area.* The region of the impulse.

(4) *The tricuspid area.* The region of the fourth and fifth left costal cartilages, close to the left edge of the sternum.

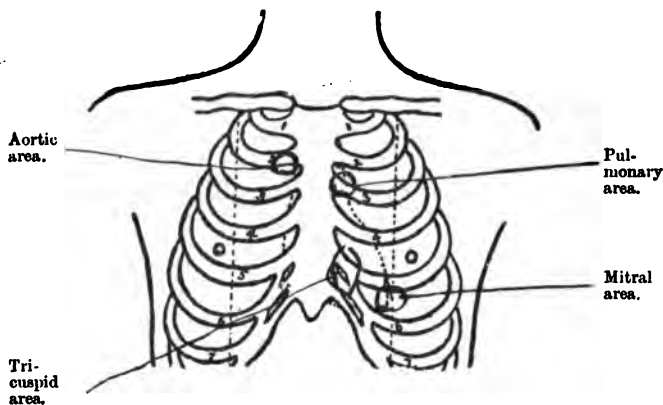


FIG. 2. A diagram to show the clinical surface-markings for the auscultation of the cardiac valves.

The mitral valve has two flaps, one, the larger, which is situated to the right and in front, lies between the auricular and aortic openings, and is called the aortic flap of the mitral. It is the flap which is concerned in the production of the aortic presystolic murmur.

The three flaps of the tricuspid valve are placed one in front, between the conus of the pulmonary artery and the auriculo-ventricular opening; one to the right, and one posteriorly, against the ventricular septum in the region of the undefended space.

The three cusps of the aortic valve are placed one anteriorly and two posteriorly, and the two coronary vessels are given off immediately above the anterior and left posterior valve.

The pulmonary cusps lie two anteriorly and one posteriorly.

6. THE INNERVATION OF THE HEART

The heart is innervated by the branches from the pneumogastric named the superior, and inferior cardiac nerves, and by branches from the superior, middle, and inferior cervical ganglia of the sympathetic.

These meet in the cardiac plexus, which is subdivided into :

(1) The *Superficial cardiac plexus* situated in the hollow of the aortic arch, superficial to the pericardium. Among other branches, this supplies a right coronary branch and one to the pulmonary plexus.

(2) The *deep*, far larger cardiac plexus, lies behind the arch of the aorta, and is again divided into right and left parts. The right supplies the right and the left the left coronary plexus.

The cardiac nerves lie in the furrows of the heart, are imbedded in the myocardium, and are supplied with ganglia, especially in the interauricular septum, and at the auriculo-ventricular junction.

7. THE RELATIONS OF THE THORACIC AORTA

The aorta arises from the left ventricle behind the conus of the right ventricle, and has as an anterior relation the left half of the sternum at the level of the third costal cartilage. On the right side immediately in front of this part of the vessel lie the pulmonary artery and right auricle. For about

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two inches the aorta is enclosed in the pericardium, and in this part are situated the sinuses of Valsalva which are the usual sites of an intrapericardial aortic aneurysm. Passing upward and to the right it lies close behind the right margin of the sternum, with the superior vena cava to the right, the pulmonary artery to the left, and the left auricle, right bronchus, and right pulmonary artery behind. From the anterior sinus arises the right, and from the left sinus of Valsalva, the left coronary artery.

At the level of the second right costal cartilage, the arch of the vessel commences and passes upward, leftward, backward and downward, round the trachea to the lower limit of the body of the fourth dorsal vertebra.

The arch lies close behind the manubrium sterni covered on each side by the pleura and lung. Important anterior relations are the left vagus and phrenic nerves, and the superficial cardiac nerves, more posteriorly the left superior intercostal vein. The left recurrent laryngeal nerve winds round the vessel. Above lies the left innominate vein. Behind lie the deep cardiac plexus, the trachea, the thoracic duct and the oesophagus. In the *concavity* of the arch lie the superficial cardiac plexus, and the bifurcation of the trachea, with the left bronchus.

From the convexity are given off from right to left the innominate, left carotid, and left subclavian arteries.

The descending thoracic aorta extends from the lower margin of the fourth dorsal vertebra to the last dorsal vertebra, and inclines from above downward, and slightly to the right to reach the middle line.

In front lie the root of the left lung and the pericardium, behind lies the vertebral column, and to the right the vena azygos major, oesophagus and thoracic duct; to the left, the left pleura and lung.

PHYSIOLOGY

A few points of physiological importance will require a brief consideration.

1. **The Cardiac Sounds.** *The first sound* is produced by such various elements as the sudden tension of the cardiac muscle, the vibration of the auriculo-ventricular valves and of the blood.

The second sound is also complex in origin, and results in part from the vibration of the sigmoid valves and in part from the vibration of the arterial walls.

2. **The Cardiac Cycle.** The two cardiac sounds form the landmarks in the cardiac cycle, which consists of:—

- (1) The auricular diastole.
- (2) The auricular systole.
- (3) The ventricular systole.
- (4) The ventricular diastole.

(1) During auricular diastole the blood streams into the auricles on account of the positive pressure in the veins aided by the elastic traction of the lungs upon the relaxed auricular walls.

(2) The auricles contract from the appendices, toward the ventricles, and the blood is forced into the ventricles. This auricular contraction is followed by a stasis of the circulation in the large veins and a floating upward of the auriculo-ventricular valves.

(3) The contraction of the ventricles is not quite complete and some residual blood is left in the cavities. With the systole the auriculo-ventricular valves are tightly locked and when the pressure exceeds the peripheral, the sigmoid valves open.

(4) With ventricular diastole the sigmoid valves close, and the auriculo-ventricular open.

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The time relations of the chief events in the cardiac cycle are as follows :—

The auricular systole lasts 0.1 of a second.

The ventricular systole „ 0.3 „ „

The auricular diastole „ 0.7 „ „

The ventricular diastole „ 0.5 „ „

3. The Cardiac Nerves are, as already stated, derived from the vagus and sympathetic.

(1) The vagus is the inhibitory nerve. The afferent nerves of this reflex are purely vagal and run in the vagus nerve to the medullary centre. The efferent nerves are probably derived from the accessory nucleus, and subsequently join the vagus.

By some the *inhibitory* centre in the medulla is supposed to be in constant tonic action, by others it is thought only to be called into action by some stimulus.

(2) The sympathetic system supplies the *accelerator fibres* to the heart; these increase not only the rate, but also the force of the cardiac beat.

The afferent fibres of this reflex pass probably by the inferior cardiac branch to the spinal cord and eventually reach the medulla oblongata, where they end almost certainly in an accelerator centre. The efferent pass from this centre and reach the inferior cardiac nerve by the annulus of Vieussens.

These cardiac reflexes have very wide connexions with the other organs of the body and in particular with the abdominal viscera.

4. Respiratory movements and the circulation.

The normal respiratory movements aid the circulation; inspiration hastens the venous and lymphatic flow, and thus favours diastole, and expiration hastens the movement of the blood into the arterial system and aids systole. The

elastic traction of the lungs by its action on the left auricle hastens the flow from the veins into the auricle, but on the other hand has little influence upon the right ventricle.

In extreme respiratory movements the effects are somewhat different. Thus, in extreme expiration the heart is small during diastole and as a result the systole is small and the pulse small. Under these conditions the elastic traction of the lungs is at its minimum.

In extreme inspiration the elastic traction of the lungs is at its maximum, the auricular contraction is embarrassed, the heart greatly distended, and the pulse small.

5. **The heart-beat.** (1) The spontaneous rhythmic action of the heart has been attributed to *automatic centres* located in the *ganglia*, the stimulation of these centres taking place in a regular order from the dominant centre situated in the auricular region. According to this view, in order to account for the fact that the contraction of the normal heart invariably starts in the great veins at their entrance to the auricle, it is supposed that the nerve-centre in this region is more irritable than that in the ventricular region.

It is, however, apparent that the ganglia are not essential for the contraction of the cardiac muscle, because the power still remains after the ganglia have been removed.

(2) Recently the view has gained ground that the automatism of the heart rests with the *muscle* itself, and the regular sequence of auricular and ventricular contraction is ascribed to direct conduction by muscle of the wave of contraction from auricle to ventricle across the auriculo-ventricular junction. *The A. V. bundle of His* (vide p. 5) is looked upon as this means of conduction.

A good deal of experimental work has been done upon this subject in order to elucidate the occurrence of '*heart-block*'.

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'*Heart-block*' is looked upon by those who uphold the conduction of the cardiac wave of contraction by the A.V. bundle as a condition in which, owing to some defect in this structure, the wave of contraction from the auricle is stopped or blocked. This heart-block may be partial or complete, and various degrees of this arrest may take place both in disease and in experiment.

Erlanger has recently stated that by means of a carefully devised clamp he has been able to graduate pressure upon the auriculo-ventricular bundle, and in this way obtain *various degrees* of heart-block. Thus only every other auricular wave may be conducted, or, with more pressure, only every third or fourth wave may produce a ventricular contraction. When there is complete dissociation, the ventricles, which are spontaneously rhythmical, commence to beat independently.

The evidence at present forthcoming upon these points is hardly sufficient to admit of a conclusive statement, but those who desire to inquire further into these interesting points should consult the writings of Gaskell, His, Mackenzie, Gibson, Erlanger, and others who have devoted particular attention to this subject.

EXPERIMENTAL CARDIAC PATHOLOGY

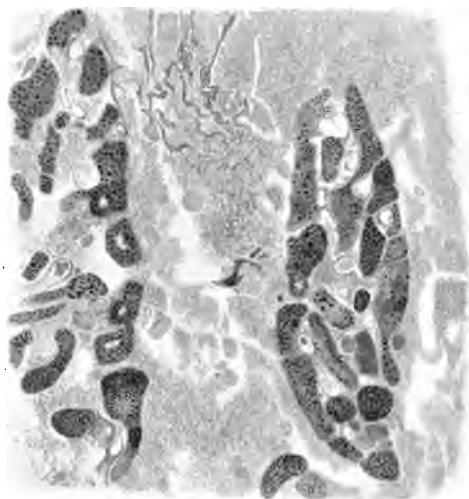
This subject has made such strides that it is necessary to make a few brief allusions to some of the important facts that we are learning from this branch of investigation.

1. The work of Stannius, Gaskell, His, Erlanger, and many others upon the *cardiac rhythm* has just been touched upon.

2. Another important branch is the exact study of *myocardial disease* by the inoculation of toxins or bacterial



PLATE II



A

A section through a portion of the left ventricle of the heart of a monkey. Death on the fifth day from rheumatic carditis. Showing fatty change in the muscular fibres. (Poynton and Shaw.)

By permission of the Pathological Society of London.

To face p. 15]



B

A section through the heart-wall, endocardium, and base of the mitral valve of a rabbit. Death from the diplococcus infection, pericarditis, and polyarthritis were present. To show invasion of the valve by the 'diplococcus rheumaticus'. (Poynton and Paine.)

By permission of 'The Lancet'.

Transverse section of
muscle.

The endocardium.

Diplococci.

The base of the mitral
valve.

cultures. Flexner and Sydney Martin's early work has been followed by many further investigations, and recently Dudgeon has published some useful facts which illustrate how much is to be learnt from this method of research. Working with the diphtheria intra-cellular toxins he has shown that experimental fatty changes may be detected in sixteen hours. The rapidity with which fatty changes may occur from bacterial toxins is indeed surprising. Thus, when working with Paine, I found the 'diplococcus rheumaticus' produced such changes in under a *fortnight*, and with Vernon Shaw, that in a monkey, dead on the *fifth* day from the same infection, such changes were also present in the myocardium.

Dudgeon, again, records an interesting fact when he finds that the *diphtheria antitoxin*, freely injected, appears to antagonize the poisonous action of the toxins on the cardiac muscle in rabbits.

3. For many years *experimental endocarditis* has been occasionally produced by investigators, and now the rheumatic infection, by reason of its frequent tendency to attack the heart, has enabled a good many definite facts to be ascertained which could formerly only be suspected.

Thus (1) every stage between simple and malignant endocarditis can be produced by one micrococcus. This alone, as will be obvious to the reader later, greatly simplifies the study of valvular lesions.

(2) Fatal dilatation, without pericarditis or endocarditis and with ante-mortem thrombosis, may result from experimental infection, thus proving the importance of the cardiac dilatation in rheumatic and other forms of heart disease.

(3) A much more accurate idea is obtained of the rapidity with which vegetations form in heart disease. In

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three days, for example, an obvious lesion may be visible on the mitral valve of a rabbit.

(4) White infarcts again may be detected in the kidney when there is no sign of a valvular lesion.

(5) The origin and mode of formation of valvular lesions and pericardial exudations can be accurately studied and the various stages traced.

These are some of the points which are made clearer by a study of experimental carditis.

4. The difficult problem of *cardiac dropsy* is being elucidated, and among the most recent researches, those of C. Bolton tend to show that the mechanical factor in its causation is not so prominent as clinical observation alone might lead one to suppose.

5. Another interesting line of experimental research is that upon *diseases of arteries*. In 1903 Josué produced arterial lesions in rabbits by the intravenous injection of adrenalin, and this fact has been amply confirmed. Fischer also produced lesions with digalen, and Boinet and Romary and Klotz have produced arterial lesions with the 'streptococcus' and bacillus typhosus.

Paine and I found inflammation and calcification of the first part of the aorta in a rabbit which had been intravenously inoculated with the diplococcus of rheumatic fever three weeks before, and which was manifesting rheumatic arthritis at the time it was killed. This again, when the clinical relationship of rheumatism and arterial disease is remembered, is at least suggestive.

It would be out of place here to enlarge upon this side of the study of heart disease to any greater length, and these brief statements are only intended to show how distinctly the accurate pathology of heart disease is advancing.

PLATE III



A

Two chordae tendineae attached to the mitral valve of a rabbit dead of carditis. The lower border of the mitral valve is just visible. On the left chorda are two inflammatory swellings, both the result of the infection. (Poynton and Paine.)

By permission of 'The Lancet'.



B

Experimental malignant endocarditis of the mitral valve (rabbit), produced by the diplococcus rheumaticus. (Poynton and Paine.)

By permission of the Royal Medico-Chirurgical Society.

CHAPTER II

THE GENERAL PRINCIPLES OF CLINICAL INVESTIGATION IN HEART DISEASE

Schema in three divisions. *Div. I*: Onset of illness—Duration of—Symptoms of—Pain—Dyspnoea and cough—Cheyne-Stokes respiration—Palpitation—Syncope—Digestive disturbances—Nervous symptoms—Wasting—Oedema—Personal history—Family history. *Div. II. Sect. 2*: Heart—Inspection of—Palpation—Percussion.

THIS and the two following chapters are devoted to the elements of clinical investigation in heart disease considered *generally*, and not with reference to any particular disease.

A methodical procedure is essential, for otherwise it is exceedingly difficult to grasp the main facts of the illness, and indications that are apparently unimportant, but which are in reality of the greatest value, may thus be easily overlooked.

There are numerous clinical schemes in use, and the one that is given here is in no sense elaborate, but will, it is hoped, prove of sufficient detail to be of service to those who are commencing the study of the subject.

A SCHEME FOR INVESTIGATION OF CASES OF HEART DISEASE

Division I. The investigation of the history of the illness. This includes an inquiry into:—

1. The onset, duration, and symptoms of the present illness.

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2. The personal history.

3. The family history.

Division II. The investigation of the cardio-vascular system.

1. The examination of the pulse with reference to :—

a. Rate.

b. Rhythm.

c. Character of wave.

d. Condition of arterial wall.

e. Special peculiarities.

2. The examination of the heart by the methods of :—

a. Inspection.

b. Palpation.

c. Percussion.

d. Auscultation.

and in some cases by—

e. Radiography.

3. The examination of the other blood-vessels, viz. :—

a. Arteries.

b. Capillaries.

c. Veins.

Division III. The investigation of other organs of the body in order to determine the amount of secondary damage that has resulted from the primary heart disease.

Attention should be directed particularly to

The condition of :—

a. The liver, stomach, and intestines.

b. The lungs.

c. The urine.

d. The connective tissues, with a view to the occurrence of dropsy.

e. The occurrence of embolism, thrombosis, and infarction.

INVESTIGATION IN HEART DISEASE 19

With this scheme as a guide, the clinical manifestations of heart disease can now be considered *seriatim*.

DIVISION I. THE INVESTIGATION OF (a) THE ONSET, (b) DURATION, AND (c) SYMPTOMS

(a) A short experience will show what wide differences there are in the manner of *onset* of the various cardiac affections. The commencement may be abrupt, as when, for example, an aortic valve is ruptured from over-exertion; or acute, as in severe rheumatic pericarditis; or exceedingly gradual, as in the degenerative lesions of the endocardium.

(b) The *duration* of the symptoms also differs greatly. Organic disease may be of long standing, and yet no complaint be made by the patient, because the condition has been compensated by hypertrophy of the heart. In other cases there is the history of repeated attacks of cardiac failure, and due weight must be attached to this evidence of slight reserve power. Yet again, urgent symptoms may date from some shock, accident or severe illness, and in some cases there may be a history of the congenital origin of the cardiac symptoms.

(c) The *symptoms* of heart disease are often of the greatest clinical importance, and long experience is required to estimate them at their proper value.

A general rule can be formulated to this effect:—

That, firstly, in *degenerative* heart affections, symptoms are of more value than physical signs.

Secondly, in *functional* heart affections, symptoms are of less importance than physical signs.

Thirdly, in *chronic valvular* disease, the value of the symptoms and physical signs is more equally balanced.

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Another point to be emphasised in considering symptoms, is the distinction between those which are the result of the *primary* condition producing the heart disease, be it rheumatism, influenza, overstrain, or other cause, and those which are the direct cause of the *heart disease itself*. In the chapters upon rheumatic affections, the importance of this distinction will be readily recognized.

The symptoms of morbus cordis are numerous, especially if we take into account those that are the result of secondary damage to other viscera. Here only the most frequent and distinctive will be mentioned. The less usual ones will be dealt with under the diseases in which they occur.

Among the most important are :—

1. Pain, precordial sensations, and tenderness.
2. Dyspnoea and cough.
3. Palpitation.
4. Syncope.
5. Digestive disturbances.
6. Mental changes, such as sleeplessness, vacillation of purpose, nervousness.
7. Wasting.
8. Oedema.

1. Pain is a frequent symptom. The most characteristic and terrible example is *angina pectoris*. Severe pain may also occur in *acute pericarditis*, but it varies much, being dull, shooting, burning, or stabbing in character. *Over-distension of the heart* gives rise to great suffering, and in *aortic disease* pain is usually a feature towards the end of the illness. Yet, in spite of these examples, it may be affirmed that organic heart disease is often singularly free from severe pain, and that its occurrence is frequently more suggestive of functional affections than of definite structural defect.

More frequent in their occurrence are *precordial sensations*,

which are variously described as 'a sense of weight', an 'all gone' feeling, or merely as a sense of uneasiness in the region of the heart.

Under the disease 'angina pectoris' allusion will be made to the widely-spread referred pains and tenderness met with in that affection. Mackenzie and Head have shown that in other cases of cardiac pain associated with tenderness, the area of this tenderness extends in front from the second rib to the horizontal nipple level, and postero-laterally from the level of the seventh cervical to the fourth dorsal spine; together with these there are others upon the inner aspects of the arms.

In some instances there may be a point of extreme tenderness over the second left costal cartilage at its junction with the sternum.

2. **Dyspnoea and Cough.** There are three forms of dyspnoea in heart disease :—

(1) Dyspnoea on exertion.

(2) Persistent dyspnoea.

(3) Paroxysmal dyspnoea.

(1) *Cardiac breathlessness* shows itself first on *exertion*, and may be met with in all forms of heart affection. It is, however, in uncompensated mitral conditions that the most severe dyspnoea occurs, for then it has a twofold origin, in part *cardiac* and in part *pulmonary*. The breathlessness of heart disease is a striking clinical symptom, for it may be intense and yet there may be no obvious changes to be discovered in the lungs. When the disease is advanced, the least exertion may cause this dyspnoea, and alteration of position may produce an intense feeling of suffocation.

(2) Later still, *persistent dyspnoea* supervenes. In many cases the patient is unable to lie with the head low, and is compelled to be propped up in some semi-recumbent position,

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or even is obliged to sit up in a chair. To this phenomenon is given the name of *orthopnoea*.

The influence of gravity upon the venous circulation of the head and neck takes a prominent part in this form of cardiac dyspnoea, for when the head is low the respiratory centres tend to be poisoned by the congestion of venous blood, the circulation of which is no longer aided by gravity.

There are, however, other factors to be considered in the production of cardiac dyspnoea. One of the most important is *disturbance of the nervous equilibrium* that normally exists between the cardio-vascular and the respiratory apparatus.

In the healthy, increased exertion means increase in the cardiac energy, and a corresponding increase in the respiratory movements. In heart disease this equilibrium is impaired, for increased exertion is not properly responded to by the damaged heart, and the respiratory and vascular responses to exertion are therefore not in accord with one another.

Then, again, the investigations of von Basch point to another factor. Distension of the capillaries in the lungs from high pressure in this system produces a *rigidity* of the *walls of the alveoli*; a condition, in fact, comparable to the changes that occur in erectile tissues. As a result of this rigidity, the inspiratory and expiratory movements must be increased in order to obtain efficient aeration of the blood.

Anaemia, such as occurs in aortic disease, must also aid in producing dyspnoea, and when there is advanced arterial disease affecting the cerebral vessels, the blood supply to the *medullary centres* may be impaired by the diminution in the lumen of the vascular channels as a result of endarteritis obliterans. *Pleural effusion*, a secondary result of the diseased heart, must always be remembered as a possible explanation of the dyspnoea.

(3) The *paroxysmal type*, or cardiac asthma, should suggest the complication of *renal disease*, either primary, or secondary to arterio-sclerosis. This disease may be the primary illness, and the heart condition secondary to it, or, on the other hand, the renal disease may have supervened in the course of the cardiac disease.

This form is frequently *nocturnal*, and is often disproportionate to the affection of the heart. The intensity of breathlessness may be extreme, and the condition is one most detrimental to recovery.

Another cause for the paroxysmal type of dyspnoea is the combination of heart disease *with true asthma*. In such cases, the congestion of the pulmonary circulation favours the occurrence of bronchial spasm.

Cough. In some cases of pericarditis cough is peculiarly irritating and distressing, and in aneurysm pressure upon the trachea or a main bronchus may alter the timbre of the cough to a metallic character.

Yawning may be continual and distressing in dilatation of the heart.

Cheyne-Stokes breathing is characterized by periodic variations, both in the extent and rate of the respiratory movements. During one phase they become increasingly rapid and ample, and then die down to extinction. A pause of complete apnoea follows, to be in turn replaced by a repetition of the rising and waning respiratory movements. According to the latest investigations, it would appear that this periodicity is in great part due to a *diminished excitability of the nervous system* brought about by a defective supply of arterial blood. Taylor, Pitt, Pembrey, and French have shown that the stage of apnoea, which they attribute to a lack of sufficient carbon dioxide to stimulate the nerve-cells, can be abolished by the inhalation of air containing more

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than 2 per cent. of carbon dioxide, or by giving pure oxygen. Cheyne-Stokes respiration is sometimes a well-marked feature in cases of dilatation of the heart resulting from renal disease. It is a symptom of very serious importance, but may be present for weeks, and yet eventually disappear.

3. **Palpitation.** This is a sensation which gives the patient the impression that the heart is acting with great frequency and violence. In many instances this is really the case, but it is not invariably so, for there may be no apparent increase in the energy or frequency of the heart's action, and yet the sensation of palpitation be present. The distressing feature in palpitation is the sense of tumult within the breast. Mere rapidity of action may not give rise to any discomfort, and the patient is sometimes quite ignorant of its existence.

Palpitation is essentially a nervous symptom, and occurs more frequently in females than in males. There may or may not be serious heart disease. This symptom will be considered in more detail in the chapter upon neuro-muscular affections of the heart.

4. **Syncope.** This alarming symptom may be the abrupt termination of a case of organic heart disease, and it is *aortic disease* and the conditions due to *degenerative lesions of the cardiac muscle* that are the most likely to end in this way. Mitral lesions, on the other hand, do not as a rule incur this danger if the myocardium is sound.

An attack of syncope, however, far more frequently points only to some *temporary and functional derangement* of the compensatory power of the heart. Reflex disturbances whether external or internal, which in the normal state are met by the heart without the least difficulty, are, under these circumstances, sufficient to reduce the force of the cardiac systole almost to the vanishing point.

The symptoms of a fainting attack are well known, but it is a far more difficult matter than might be thought, to distinguish between a fainting attack and some of the *minor epileptiform seizures*. In fact the diagnosis may be impossible, for some epileptiform seizures are cardiac, and produce a condition of syncope; and, on the other hand, some syncopal attacks are as abrupt as any 'petit mal'.

Inquiry should be made into the nature of the attack, and importance must be attached to the premonitory symptoms of giddiness, sickness, and sinking. The patient, in an attack of syncope, breaks out into a clammy sweat, the colour of the face turns to a greenish pallor, the sight grows dim, surrounding objects swim round, and finally in the complete faint consciousness is lost. The pulse at first fails, and the cardiac sounds are almost inaudible. The loss of consciousness is usually momentary, and the recovery gradual and distressing, but in the graver forms of heart disease, the period of unconsciousness may be much prolonged.

5. Digestive Disturbances. There are few symptoms that are of greater practical importance than these. They may be *early warnings* of cardiac failure, and they are repeatedly a source of great trouble to the patient and to the physician during the course of chronic valvular disease.

They must be studied from two points of view:—

1. The influence of disordered cardiac action upon digestion.
2. The influence of disordered digestion upon a damaged heart.

(1) An impaired or vitiated blood supply to the alimentary organs soon produces lack of tone, and, as consequences, *delayed and imperfect digestion*. When there is a breakdown of the right side of the heart, these consequences are

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exaggerated, and the liver, pancreas, stomach, and intestines are all engorged with imperfectly aerated blood.

Flatulence, constipation, loathing of food, pains between the shoulders, oppression after meals, and vomiting, are among the most frequent complaints.

(2) On the other hand, an impaired digestion reacts unfavourably upon the damaged heart by depriving it of a full supply of nutrition. Distension of the stomach or transverse colon by upward pressure upon the embarrassed right ventricle may produce the most dangerous cardiac failure, and for this reason in some of the *paroxysmal affections* of the heart, notably angina pectoris, dyspepsia may be a symptom of the gravest significance.

6. Nervous Symptoms. Both in childhood and in adult life, organic heart disease may produce considerable nervous disturbance and even insanity. Children, particularly when they *suffer from aortic disease*, are easily terrified, much worried by lessons and easily excited by companionship. The business man loses his grasp of figures, or his nerve in organization.

These and other similar warnings must be differentiated from those mental symptoms, that are the result of the *dread of heart disease*, either fancied or real. There is no doubt that much mental suffering may be caused by undue stress being placed upon functional symptoms by a medical man, and that in many cases of organic disease the patient is happier and better with information that is not made too explicit.

7. Wasting. Failure of general nutrition is a very important sign of organic heart disease in childhood. It is met with at all ages, but in children particularly it is an early and striking feature.

8. Oedema. This symptom, again, is one of great frequency

and importance. It shows itself in the most dependent parts, and is generally first noticed by the patient at the end of the day as a puffiness around the ankles. In childhood there may be for some unexplained reason oedema of the face, and an appearance most suggestive of kidney disease.

The occurrence of oedema, and later, dropsy, is so closely bound up with failure of compensation, that its fuller consideration is deferred until the study of the secondary effects of heart disease.

THE PERSONAL HISTORY

of the patient may be very helpful. Rheumatic fever—a most frequent cause of heart disease—for example, often attacks the individual more than once. Other diseases, notably syphilis and influenza, may also grievously damage the heart. Poisons, such as alcohol and, to a less degree tobacco, are also detrimental. Lastly, occupation and idleness have their significance.

The stvedore who is continually exposed to sudden and excessive exertion may develop an *aneurysm*. The well-to-do business man, harassed by the anxiety of heavy speculations, too well fed and too sedentary, may fall a victim to *angina pectoris*. The stout, middle-aged lady meandering through life in her carriage, with few worries and less exercise, may become the possessor of a *dilated and fatty heart*.

THE FAMILY HISTORY

may be of especial value, in those most difficult cases of myocardial weakness in the elderly, for which no obvious cause is apparent, for a history of heart disease and fatal syncope may be elicited in several near relations, which will

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at once place the physician on his guard as to the interpretation of the particular case upon which he is consulted.

Following the schema, the next step would be the *examination of the pulse*, but there are many points in its study which are more easily understood after the heart has been considered. On this account, the order of the schema will be departed from, in so far, that Section 2 of Division II will be taken before Sections 1 and 3.

DIVISION II. SECT. 2. THE EXAMINATION OF THE HEART

a. Inspection

Much that is of value can be learnt by this method, which should include more than an inspection of the precordial area.

The *general appearance* of the patient should be noted.

The aspect may be *pale* and anxious as in some cases of aortic disease, or *cyanotic* as in congenital heart disease or advanced mitral affections. The conjunctivae may be *icteric* and the skin sallow when the liver is greatly congested from back-working.

The extent of *dyspnoea* can be gauged, and the general nutrition of the patient ascertained.

Brisk friction of the skin of the forehead may reveal the *capillary pulsation* of aortic regurgitation.

The local examination consists in the inspection of:—

1. The precordial area.
2. The neck.
3. Abdomen and extremities.

1. *The precordial area.*

(a) The chest may be bulged forward by a large hypertrophied heart; in childhood this is sometimes a very striking feature.

(b) A more important observation is that of the position and character of the impulse. Is it localized or diffuse?

Does the precordial area heave as in *hypertrophy*, and is the impulse displaced downward and outward, or is the impulse only just visible, and displaced outward as in *dilatation*?

In some cases of *adherent pericardium*, the lower part of the sternum and adjacent intercostal spaces are retracted in the systole.

The whole heart may be displaced by a *pleural effusion*, and an impulse be visible only to the right of the sternum, or far in the left axilla.

Attention must be directed to *epigastric pulsation*, and lastly to any abnormal pulsations as in *aneurysm*.

Enlarged *thoracic veins* will be noted.

2. Inspection of the neck will detect the wild throbbing of severe *aortic regurgitation*, and the engorged and sometimes visibly pulsating jugulars of *tricuspid incompetence*.

3. Inspection of the abdomen may lead to detection of *ascites* or of an *enlarged liver*, and lastly, examination of the extremities may discover *oedema*, and pressure on the quick of the finger-nails, demonstrate *capillary pulsation*.

b. Palpation

The whole hand should first be laid upon the chest, and then the impulse or any abnormal pulsation accurately localized with the *tips of the fingers*.

This method is especially valuable for ascertaining the exact position, and estimating the strength of the *cardiac impulse*.

In arriving at any conclusions upon this point, particular attention must be directed to the configuration of

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the chest, and to the absence or not of hypertrophic emphysema; the results that are obtained must be compared with the pulse, and thus the error is avoided of mistaking a normal heart, the impulse of which is almost or quite impalpable on account of emphysema or other extra-cardiac causes, for a dilated or feeble one.

When the left ventricle is *hypertrophied*, the impulse is displaced downward and outward, and is forcible and heaving. Hypertrophy of the right ventricle produces a powerful heave in the epigastrium. When the left ventricle is *dilated*, the impulse is feeble and tapping, and displaced outward, and in some cases no impulse may be detected at all.

Should, as is so often the case, both dilatation and hypertrophy be present, their relative prominence may be estimated in some degree by this method of palpation. In all cases a note should be made of the *extreme limit to the left*, at which the impulse is palpable, and the maximum of the beat not alone relied upon.

Should the pulmonary or aortic second sound at the base be greatly *accentuated*, the shock at the closure of the valve can be distinctly felt by the hand.

A physical sign of great value is the *diastolic shock*. This is most frequently detected in the upper part of the chest, over an aneurysm. In time it follows immediately upon the true impulse, and is ascribed to the recoil of the aortic wall after its systolic distension.

Valuable evidence is also obtained from palpation by the detection of *thrills*.

These thrills give a sensation which has been compared to that which is felt when the hand is laid upon the back of a purring cat.

Their mechanism is further considered under the study

of the cardiac murmurs ; here it will suffice to say that they are produced by a strongly acting heart, causing vibrations in the blood current, at points where fluid veins are formed.

These thrills are divided, according to their position in the cardiac cycle, into :—

- | | |
|----------------|------------|
| 1. Presystolic | } Thrills. |
| 2. Systolic | |
| 3. Diastolic | |

1. (a) The *presystolic thrill* occurs more frequently than any other, and its detection is of most valuable assistance in the diagnosis of mitral stenosis. The presystolic thrill in such cases is felt over a limited area in the region of the impulse.

(b) In tricuspid stenosis, a very rare affection, a *presystolic thrill* may also be detected. This is localized to the region of the fifth chondro-sternal junction on the left side.

(c) In some cases of aortic regurgitation a *presystolic thrill* is produced at the impulse. The explanation of this is difficult ; some believe that it is produced by the regurgitant aortic stream setting up vibrations by its impact on the anterior flap of the mitral valve ; others that this flap is unable to retract completely on account of the reflux of blood into the left ventricle, and that in this way a narrowing of the mitral orifice is produced, and the thrill then produced by the contraction of the left auricle.

2. The *systolic thrills* are classified into :—

(a) Mitral ; (b) Aortic ; and (c) Pulmonary.

(a) The mitral thrill is palpable over the impulse, and indicates *mitral regurgitation*. This thrill is not very common.

(b) The aortic thrill is localized to the base of the heart and large vessels of the neck, and usually indicates *aortic stenosis*.

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(c) The pulmonary thrill, with its maximum in the second and third left intercostal spaces, indicates *pulmonary stenosis*.

A systolic thrill may also be detected over the upper part of the chest in some cases of aneurysm of the thoracic aorta.

3. *Diastolic Thrills*. These are almost invariably results of *aortic regurgitation*, and may then be palpable over a wide area, extending from the aortic cartilage downward. Very exceptionally a diastolic thrill indicates *pulmonary regurgitation*.

Pericardial and pleuropericardial friction occasionally give rise to a palpable fremitus, but this is only confirmatory of much more trustworthy auscultatory evidence.

There are many instances in which palpation must be extended from the precordial area to other parts of the chest, to the neck and abdomen. A few examples will be sufficient for illustration.

Pulsation may be discovered in abnormal situations as the result of an *aneurysm*. A thrill, too, may be felt in the large arteries at the root of the neck, or the liver may be found to be pulsating.

Tracheal tugging is a particularly interesting example of the value of palpation. This occurs most characteristically when an aneurysm of the transverse part of the arch of the aorta is adherent to the trachea or a main bronchus. With each pulsation the trachea is dragged upon by the aneurysm, and if the patient's head is thrown back, the fingers placed on the lower rim of the cricoid cartilage, on either side of the middle line, and the larynx then *lightly pressed upward*, a definite tug or jog is detected with each cardiac pulsation. The normal pulsation of the thyroid arteries must not be mistaken for this phenomenon.

c. Percussion

For percussion, no instruments are comparable to the fingers, for only by these can the two essential elements in this method be fully recognized.

(1) Tone.

(2) Resistance.

Percussion should be light even when it is firm, and these precautions should be observed.

1. Percuss from the wrist.

2. Percuss on to the region between the base of the nail, and terminal finger-joint.

3. Let the percussing finger fall vertically.

A.—*The extent of the relative or deep cardiac dulness.* This is the most important result of percussion. It is often difficult, and sometimes impossible, to ascertain these limits, but there is no doubt that a very high degree of accuracy can be obtained if the beginner will commence by marking the outline on the chest. Though laborious, this produces a confidence which is later invaluable.

A *definite routine* is best adopted. A convenient one is to begin from above and mark the upper limit, then the left, and lastly the right. It is rarely necessary to define the lower limit.

It must be emphasized that unless the heart is displaced, the deep cardiac dulness invariably extends beyond the *right margin* of the sternum.

B.—*The superficial or absolute cardiac dulness* is of less importance, and often gives more information about the condition of the left lung than the heart.

The normal limits of this triangular area are approximately—(1) the mid-sternal line; (2) a horizontal line extending from this to the fifth left costo-chondral junction;

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and (8) an oblique line also drawn from the mid-sternal line : at the level of the fourth costal cartilage passing down and leftward to join the horizontal line at the fifth costo-chondral junction.

Percussion also gives information upon the occurrence of any abnormal dullness in the chest, such, for instance, as may be met with in many cases of *aneurysm* of the thoracic aorta.

The *sensation of resistance* below the pleximeter finger must be carefully noted, for it is often a guide in the estimation of the limits of the heart. This feeling of resistance reaches its maximum over a large pericardial effusion.

Ortho-percussion. Goldschneider in 1905 directed attention to this method as the most accurate when tested by orthodiagraphy. It can, however, only be used when there is absolute silence in the room. The technique is the following. The tip of the percussed finger alone touches the chest, and the terminal phalanx is flexed as far as possible upon the second. Percussion is made upon the terminal inter-phalangeal joint, and the terminal phalanx is not kept at a right angle to the surface of the chest wall, but parallel to a line perpendicular to the sternum. The percussion adopted is so light that the ear must be placed close to the chest wall, and the attention first directed to the note which gives the minimum of resonance when the percussion is made over *the lung alone*. Directly the finger comes to the limit of the cardiac dullness, this resonance will be replaced by a completely dull note.

CHAPTER III

THE GENERAL PRINCIPLES OF CLINICAL INVESTIGATION (*continued*)

Div. II. 2: Auscultation—The cardiac sounds—Adventitious sounds—Cardiac murmurs — The cardinal murmurs — Combined organic murmurs—Murmurs due to relative incompetence—Mechanics of the heart in valvular lesions—Multiple valvular murmurs—Functional murmurs—Cardio-pulmonary murmurs—Disappearance of cardiac murmurs—Duration of murmurs—Arterial and venous murmurs—Scheme of the various murmurs—Pericardial friction—Air and fluid in pericardial cavity—Pleuro-pericardial friction—Graphic record of cardiac disease—Radiographic examination of the heart.

THE EXAMINATION OF THE HEART (*continued*)

d. Auscultation

THE binaural stethoscope, now in general use, needs no description ; the purchaser should be careful to assure himself that the ear-pieces do not fret his auditory meatus, that the spring, if this form is chosen, is not too strong, and that the rubber tubing is thick. Lastly, the chest-piece has often a poor screw, and if this is the case it will be continually dropping out and getting lost. The beginner, should use a simple instrument, and I am not convinced of the value of the various stethoscopes used for magnifying sounds. The danger with auscultation, lies rather in hearing *too much* than too little.

In the chapter upon the anatomy of the heart, the surface-markings for the valvular orifices were indicated, and it was

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also pointed out that in the auscultatory study of the diseases of the valves, there were four important areas, namely :—

1. The mitral, with its centre at the impulse.
2. The aortic, at the inner end of the second right inter-costal space.
3. The pulmonary, at the inner end of the third left inter-costal space.
4. The tricuspid, at the fifth chondro-sternal junction on the left side.

Auscultation accordingly should commence with an investigation of these areas. This completed, the entire precordial region is examined, and in many cases also the left axilla, back of the chest, and neck must be included.

1. THE CARDIAC SOUNDS

Attention is first directed to the first and second sounds of the heart.

Changes may affect either the first or second sound, and are classified under :—

1. Alteration in length.
 2. Alteration in intensity and pitch.
 3. Alteration in rhythm.
 4. Alterations in their relationship to one another.
- (1) When the ventricles are hypertrophied, and, in spite of this, are beginning to yield to peripheral high pressure, *the first sound is lengthened.*
- On the other hand, in dilatation it may be considerably shortened. It may also be short when the ventricles are weak and yet not necessarily dilated. This sign is of much importance in fatty degeneration of the heart.
- (2) In hypertrophy, the first sound is low-pitched and dull, in dilatation, high-pitched and clear.

In estimating these changes it must be remembered that the first sound may be altered *from extrinsic causes*. A pericardial effusion, or emphysema, or adipose tissue intervening between the chest wall and the heart may, for example, muffle the sound almost to extinction.

(3) (a) *Reduplication of the first sound* is the result of a certain degree of asynchronism of the right and left ventricles. This may arise either from the two auriculo-ventricular valves not closing with exact coincidence, or possibly because the full tension of the cusps does not occur at the identical moment. This condition points to a commencing failure of the power of one or other ventricle, such as may occur in chronic renal disease and obstructive pulmonary disease.

(b) *Irregularity* of the first sound may vary from a slight alteration in time and intensity, to a most extreme degree of arrhythmia.

Irregularity of the action of the heart occurs in many forms of disease, and is a striking feature of advanced mitral disease.

(c) By *intermission* is meant the disappearance of the first sound for one beat; this may occur every few beats, and is sometimes a constant feature in people who are in apparently perfect health. On the other hand in degenerative myocardial lesions it may be a sign of serious danger.

The second sound may also show variations in duration, intensity and pitch, and rhythm.

(1) When the action of the heart is feeble and rapid, the second sound becomes shorter.

(2) When the tension of the pulmonary circuit is high, the *pulmonary second sound* at the base is accentuated and frequently reduplicated. Similarly, when the tension is high in the systemic arteries, as for example in renal disease, the *aortic second sound* may be accentuated.

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On the other hand, a diminution in the intensity and clearness of the aortic second sound may be the earliest evidence of an endocarditis of the valve.

In *childhood*, the pulmonary second sound is usually louder than the aortic ; in *adults*, the reverse is often the case.

(8) In the section on Mitral Stenosis, allusion will be made to an apparent reduplication of the second sound at the apex. Upon auscultation the cardiac sounds give the impression which is represented by the words *lub-tüt-tüt*. The double sound (*tüt-tüt*) is, if persistent, an important sign that there has been some *organic damage* to the mitral valve, which interferes with its perfect mechanism.

(4) The *relation* of the first to the second sound may be altered. In renal disease, for example, when the heart is commencing to fail before the high peripheral pressure, the systolic interval is prolonged at the expense of the diastolic, and the first and second sounds are equidistant from one another. This is termed the *tic-tac heart-beat*.

An ill-omened occurrence is an *approximation of the first and second sounds*, on account of the shortening of both the systolic and diastolic intervals. In extreme cardiac weakness, not only are these sounds short, but they approximate to one another, on account of the inefficient systole and imperfect emptying of the ventricles.

2. ADVENTITIOUS SOUNDS

These are subdivided into two classes:—

1. Cardiac, arterial, and venous bruits.
2. Friction sounds.

Class I. Cardiac murmurs or bruits. When, in the course of circulation of fluid through a series of tubes, a sudden constriction occurs at some point, there arises immediately

beyond the point of constriction a fluid vein which gives rise to vibrations in the stream. These vibrations, if sufficiently powerful, are sonorous. In the course of the circulation of the blood, constrictions may arise, and are often irregular in form, as a result of disease or imperfection of the valves. At these points, if the heart has sufficient power, sonorous vibrations result which are conducted through the wall of the heart and chest, and are recognized on auscultation as bruits or murmurs. Further, if these vibrations are sufficiently powerful, they can be recognized on palpation as thrills, q. v. This explanation of the production of cardiac murmurs is not wholly satisfactory, but it is difficult to improve upon this generally received view upon the subject.

These murmurs are classified as :—

1. Organic.
2. Inorganic or functional.
- I. The organic murmurs are subdivided in accordance with their position in the cardiac cycle into :—
 1. *Systolic*, produced by the systole of the ventricles.
 2. *Presystolic*, produced by the systole of the auricles, and synchronous with their systole.
 3. *Diastolic*, produced during the diastole of the ventricles.

GROUP A. THE CARDINAL MURMURS

For the sake of clearness, those murmurs which may be called the *cardinal* ones will be taken first, and will be considered as they occur in *valvular disease*.

The results of valvular disease are to produce :—

- (1) Incompetence or leakage of a valve.
- (2) Stenosis or obstruction.
- (3) A combination of both conditions.

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The simple lesions, incompetence and stenosis, will be first dealt with.

Either of these may occur in connexion with each of the four valves, giving rise to:—

1. Mitral incompetence	Mitral stenosis.
2. Aortic "	Aortic "
3. Tricuspid "	Tricuspid "
4. Pulmonary "	Pulmonary "

1. In *mitral incompetence* the diseased mitral valve does not close accurately in the systole of the left ventricle, consequently at that time there is a leakage of blood through the aperture into the left auricle, and a fluid vein is caused at this point. A bruit results which is *systolic* in time.

2. In *aortic regurgitation* the damaged valve closes imperfectly, a leakage must then occur in diastole and blood pass back into the left ventricle through the injured valve. A fluid vein is formed at this point. The bruit is *diastolic* in time.

3. In *tricuspid incompetence*, the fluid vein occurs during the systole of the right ventricle at the point of leakage through the tricuspid valve. The result is a *systolic* murmur.

4. In *pulmonary incompetence*, the fluid veins occur in diastole when the blood leaks back into the right ventricle; and the result is a *diastolic* bruit.

Thus from incompetence of the valves there result:—

Two systolic bruits, mitral and tricuspid in origin, and two diastolic bruits, aortic and pulmonary in origin.

Turning next to the stenotic lesions:—

1. In *mitral stenosis*, the obstruction and fluid vein occur when the systole of the left auricle forces the blood from its own chamber into the left ventricle through the narrowed opening.

This auricular systole immediately precedes the ventricular systole and the first sound, hence the bruit is *presystolic* in time. Later it will be pointed out that the murmur may be diastolic or mid-diastolic.

2. In *tricuspid stenosis* also, the bruit will be for similar reasons *presystolic*.

3. In *aortic stenosis*, the obstruction makes itself felt when the left ventricle forces the blood into the aorta through the narrow opening; and the bruit is necessarily *systolic* in time.

4. Lastly, in *pulmonary stenosis* the murmur is also *systolic*.

Thus from stenosis of the valvular orifices there result two presystolic—mitral and tricuspid in origin, and two systolic murmurs—aortic and pulmonary.

The cardinal valvular lesions of the heart thus produce *four systolic, two presystolic, and two diastolic* murmurs.

The beginner must not get confused by the terms diastolic and presystolic murmurs. Between the two cardiac sounds there are two pauses, the systolic and diastolic; the latter, for the nomenclature of murmurs, is divided into two parts, the early part the diastolic, and the late part synchronous with the *auricular* systole—the presystolic division of the long pause.

Investigation of Cardiac Murmurs

1. In the study of these murmurs, the first step is to ascertain their *time* in the cardiac cycle, and this should be done with the hand on the *impulse*.

The other points of importance in studying cardiac murmurs are:—

2. The position of maximum intensity and the direction of conduction of the murmurs.

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3. The character.
4. The duration.
5. Their relation to the cardiac sounds.

The point of maximum intensity and the direction of conduction are of great assistance in differentiating the bruits that occupy the same position in the cardiac cycle.

A. The four systolic murmurs show these differences:—

1. The mitral systolic, pointing to mitral regurgitation, has its area of *maximum intensity* in the region of the *impulse*, and can be traced into the left axilla. It is often audible over the back of the chest, and may be traced directly round from the axilla, or heard there independently.

2. The tricuspid systolic murmur pointing to tricuspid regurgitation is at its *maximum* in the fourth and fifth intercostal spaces on the left side close to the margin of the sternum. It is conducted toward the right, but is usually a soft murmur with but a slight area of conduction.

3. The aortic systolic bruit, pointing to aortic stenosis, is at its *maximum* intensity at the inner end of the second intercostal space on the right side, and is conducted upward into the vessels of the neck.

4. The pulmonary systolic bruit, pointing to pulmonary stenosis, has its point of *maximum intensity* in the third intercostal space on the left side.

B. The presystolic murmurs are usually audible over a remarkably limited area.

1. The mitral presystolic—evidence of mitral stenosis—is, as a rule, limited to the impulse.

2. The tricuspid presystolic—evidence of tricuspid stenosis—has the point of maximum intensity at the *fifth left chondro-sternal junction*.

C. The diastolic murmurs are very variable in regard to their points of maximum intensity and conduction.

1. The aortic diastolic murmur—pointing to aortic regurgitation—may be most audible,

Firstly. In the classical position over the inner end of the second right intercostal space.

Secondly. In childhood the maximum intensity is frequently in the second left intercostal space. It should be remembered a diastolic murmur in this position is much more often the result of damage to the aortic valves than to the pulmonary.

Thirdly. The maximum may be immediately over the sternum.

Fourthly. The maximum may be at the impulse.

This murmur, when the maximum is in the classical position, is often to be traced over a wide area down the right border of the sternum and upward into the large vessels of the neck.

2. A pulmonary diastolic murmur—pointing to pulmonary regurgitation—is rare, and usually a result of congenital heart disease; the point of maximum intensity is in the third left intercostal space, and it is conducted down the left margin of the sternum.

GROUP B. COMBINED ORGANIC MURMURS

The cardinal murmurs at a valvular orifice frequently occur together. (a) It is, for example, unusual for mitral stenosis to exist as a *pure* lesion, for in the process of thickening and contraction, there must almost inevitably be a certain degree of incompetence.

As a result a double murmur at the impulse, in part rumbling and presystolic, and in part blowing and systolic, is of very frequent occurrence, and points to a certain degree of stenosis and regurgitation.

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(b) An occasional occurrence is a *double murmur* in the tricuspid region—partly rumbling and presystolic, partly blowing and systolic; this points to a combined tricuspid stenosis and regurgitation.

(c) A *to-and-fro murmur* at the aortic cartilage, which can be traced down the right margin of the sternum, is also of frequent occurrence.

This murmur is partly systolic, and partly diastolic.

In some cases it points to a combination of stenosis and incompetence of the aortic valve.

(d) A *to-and-fro murmur* over the pulmonary area which is not a conducted aortic murmur is very rare, but in some cases of patency of the ductus arteriosus there may be a loud systolic and diastolic murmur.

(e) A somewhat similar bruit is also met with when an *aneurysm* of the aorta ruptures into the pulmonary artery.

In some cases in childhood there occurs a *to-and-fro mitral murmur*, the diastolic element of which is blowing and not rumbling in character. In these cases there is practically no stenosis, but an almost pure regurgitant lesion, sometimes complicated by an adherent pericardium. Again in adults, a *to-and-fro aortic murmur* may occur, and yet the lesion be in nature almost entirely regurgitant.

GROUP C. CARDIAC MURMURS THE RESULT OF RELATIVE INCOMPETENCE

These are murmurs which are often associated with organic heart disease, but *are not themselves the result of disease* of the particular valve.

Their explanation leads to a brief account of the behaviour of the heart when its mechanism is impaired by the presence of any of the cardinal valvular lesions already enumerated.

The Mechanical Results of Valvular Lesions

Firstly: *Mitral regurgitation.*

(a) In mitral regurgitation, during the ventricular systole, blood is forced back through the incompetent valve into the left auricle, and as a result this chamber receives, in addition to its normal supply from the pulmonary veins, this extra amount which is the result of leakage. The auricle dilates and hypertrophies to cope with this increase.

(b) The *left ventricle* also dilates and hypertrophies, receiving as it does a larger quantity of blood at each auricular systole, and having also to supply the system with a due quantity of blood in spite of the wastage at each systole through the incompetent valve.

(c) The stream regurgitating into the left auricle soon causes a difficulty in the *pulmonary circuit*, for the auricle forms but an imperfect barrier between the powerful left ventricle and the pulmonary circulation.

The pressure accordingly rises in the pulmonary circulation, and is met by *hypertrophy of the right ventricle*. This hypertrophy of the right ventricle is an event of very great importance, for by means of it the left auricle is supported in its struggle against the back pressure caused by the left ventricle, and when it is effectual the lesion is termed *compensated*.

(d) As the disease gains ground, the right ventricle may reach its limit of hypertrophy and commence to dilate. Should this dilatation reach a considerable degree, the tricuspid valve, though uninjured by disease, becomes, owing to the yielding of its ring, unable to close the auriculo-ventricular opening, and there now supervene *tricuspid regurgitation*, and *failure of compensation*.

This tricuspid regurgitation produces a systolic murmur in the tricuspid region, a result of *relative incompetence of the valve*,

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(e) The further steps in the process are that firstly this tricuspid regurgitation produces the same results in the right auricle as did the mitral regurgitation in the left. *The right auricle* accordingly *dilates*, and to a feeble extent hypertrophies to cope with its difficulties.

(f) Lastly, the regurgitant stream through the right auriculo-ventricular opening tells back not only on the auricle, but upon *the venous system* beyond, and the results are fullness and pulsation of the large veins, enlargement, and even pulsation of the liver, and dropsy.

2. *In mitral stenosis.* The difficulty that confronts the heart is the resistance in front of the left auricle due to the narrowed auriculo-ventricular opening.

(a) The left auricle hypertrophies to overcome this, but there is no call upon the *left ventricle*—as there is in mitral regurgitation—to hypertrophy, and later in the disease it may actually *atrophy* to some degree because the small quantity of blood it receives through the stenotic opening does not require the force of a normal systole to discharge it into the systemic arteries.

(b) As the stenosis increases the *left auricle yields*. The resistance is now felt in the pulmonary circuit, and is compensated by hypertrophy of the right ventricle. This hypertrophy is a more *prominent feature* in the history of mitral stenosis than it is even in mitral regurgitation.

(c) When the right ventricle begins to fail the development of the course of events is comparable to that described in the preceding section from (d) onwards. And here again there arises a *murmur of relative incompetence* upon the super-vention of the tricuspid regurgitation.

3. *In aortic regurgitation.* (a) The first results will be dilatation of the *left ventricle*, to accommodate the blood that leaks back through the incompetent valve during diastole,

and hypertrophy to cope with the large quantity that has to be discharged into the arterial system at each systole.

(b) Later this compensatory hypertrophy may begin to fail, and dilatation commence.

(c) Should this reach a high degree, the ring of the mitral valve is dilated, and the valve segments are unable to close the auriculo-ventricular opening. There then results a *relative incompetence of the mitral valve* and mitral regurgitation.

(d) With the supervention of mitral regurgitation there may follow all the results already detailed under that lesion, including relative tricuspid incompetence.

4. *In aortic stenosis.* There is hypertrophy of the left ventricle in order to overcome the obstruction and later there is dilatation, and then a sequence of events comparable to that described in aortic regurgitation.

The sigmoid valves, with their more rigid rings of insertion, are rarely the sites of relative incompetence, although occasionally an aneurysm may so dilate the aorta in the region of the aortic valves as to produce regurgitation, but even then the valves themselves are almost invariably damaged by the atheromatous process which produced the aneurysm.

Relative incompetence thus occurs almost exclusively in connexion with the mitral and tricuspid valves.

This incompetence may arise also from *primary dilatation of the ventricles*. Thus in simple dilatation of the heart, a systolic mitral or tricuspid murmur may appear without any organic valvular disease at all, the murmurs being the result of relative incompetence.

GROUP D. MULTIPLE VALVULAR MURMURS

It is by no means a rare event, especially in the rheumatic heart disease of childhood, to find more than one cardiac valve attacked by disease.

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By far the most important of these lesions is a combination of mitral and aortic disease. Already, under the study of the mechanism of aortic regurgitation, it has been shown that a relative incompetence of the mitral valve may arise from the dilatation of the left ventricle produced by the primary aortic disease. In some cases, however, both valves are actually damaged, and the mitral generally suffers before the aortic is attacked.

In such a case the usual result is aortic and mitral incompetence, and the two cardinal murmurs, the aortic diastolic and mitral systolic are evident upon auscultation.

Again, *mitral and tricuspid stenosis* may be associated, or *aortic stenosis and mitral disease*. In all these cases the distinctive murmurs must be carefully differentiated.

GROUP E. FUNCTIONAL CARDIAC MURMURS

These are met with most frequently in conditions of anaemia and debility. The valves are not damaged, and as these murmurs are most frequently basal, they cannot, as a rule, be classed with those which have already been referred to as results of relative incompetence.

The most satisfactory explanation of their formation is the one that ascribes them to the result of fluid veins which are sonorous; and Foxwell's explanation will be followed here to explain the most common functional bruit, the pulmonary.

All functional murmurs are systolic in time, and may be pulmonary, aortic, tricuspid, or mitral.

1. The *pulmonary*, according to Foxwell, is caused by dilatation of the conus arteriosus of the right ventricle. The ring of the pulmonary valve is thus rendered *relatively narrow*, and a fluid vein formed. In addition, the orifice of the pulmonary artery is, by this dilatation of the conus, *dragged somewhat obliquely*. The posterior half is fixed by its

attachment to the aorta, and the anterior half moves upwards in the process of dilatation. On this account the stream of blood impinges against the wall of the vessel, and may, in this way, produce sonorous vibrations.

2. The functional *aortic* murmur is explained by a relative stenosis of the valve caused by the dilatation of the ventricle immediately below.

3 and 4. The *mitral and tricuspid bruits* are probably in most cases murmurs of relative incompetence due to yielding of the auriculo-ventricular rings.

Potain is of opinion that the majority of functional murmurs are in origin *pulmonary*.

These bruits are often much altered by the *position* of the patient, rising in intensity during recumbency.

GROUP F. CARDIO-PULMONARY MURMURS

These are of the nature of functional murmurs, but are dependent upon extrinsic causes, usually pulmonary, interfering with the normal relations of the heart.

They are as a rule *systolic*. They may appear where the heart is much displaced, as in cases of severe rachitic deformity of the chest, large pleural effusions, or cirrhosis of the lungs.

Again, in some cases of mediastinitis, when the left pleura is adherent to the pericardium and the heart itself is shackled by adhesions, a systolic murmur may appear which varies with respiration.

In tuberculosis, lymphadenoma, or malignant disease, large glands may press upon the pulmonary artery and produce a systolic murmur.

These bruits are usually intensified in inspiration and fade away in expiration. The apical bruit is, at its maximum,

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internal to the apex and not actually over the impulse. There may be an interval between the first sound and the murmur, and lastly, they are modified by position, usually becoming fainter in the erect position.

THE DISAPPEARANCE OF CARDIAC MURMURS

Murmurs that are functional, or the result of relative incompetence, may vanish with the recovery of the patient.

Organic murmurs may also disappear for one of two reasons.

1. The damage may be so *slight* that there may be a true cure when the process of healing is completed. Both mitral and aortic lesions have been observed to take this favourable course.

2. In organic valvular disease, a *break-down* of the heart from myocardial weakness may be a cause of the disappearance of murmurs, and this occurs notably in cases of mitral presystolic and aortic regurgitant bruits.

As an outcome of this latter fact, it is unwise to attempt to form an accurate opinion upon a case of organic heart disease, when it first comes under observation in a condition of acute failure, for when the heart rallies, unsuspected murmurs may reappear.

THE CHARACTER OF THE VARIOUS CARDIAC MURMURS

3. The characters of these murmurs vary considerably.

The *systolic mitral murmur* is blowing, and sometimes musical; less frequently it is vibrant and gives rise to a thrill.

The *tricuspid systolic murmur* is usually soft and blowing. The *aortic systolic murmur* is often loud, harsh, and vibrant, and accompanied by a thrill. The *pulmonary systolic*

murmur also is often loud, and frequently accompanied by a thrill. In character it is generally blowing. As a rule this murmur is congenital in origin, for acquired disease of this valve is exceedingly rare. The *presystolic murmurs* are rumbling and vibrant, and usually accompanied by a thrill. The characteristic presystolic bruit of mitral stenosis rises in a crescendo to its termination in the first sound.

The *aortic diastolic murmur* is very variable in character. Usually blowing, it may be so soft that it is of all murmurs the one most easily overlooked. On the other hand, it may be so loud and musical that it may be audible to the patient, and in rare cases can be heard at a distance from the chest.

It must be remembered that a loud murmur does not necessarily mean a severe lesion. If a general rule can be formulated at all, it is that a loud murmur points usually to a slight valvular damage, and to a strongly acting heart, but there are important exceptions such as the murmur produced by a ruptured valve which is loud and yet of serious import.

THE DURATION OF MURMURS

4. Some occupy the entire interval between the two sounds, others a mere fraction of this interval.

Of more practical importance is the relation of the murmur to the cardiac sound with which it is associated. If the murmur appears to replace partially or entirely this sound, the lesion is more severe than when the sound is left intact.

Finally, with auscultation, as with the other methods, it is often necessary to investigate beyond the precordial area.

Thus a murmur may be detected in some unusual situation, as in some cases of *aneurysm* of the descending aorta. Or again, in *dissecting aneurysm*, a systolic murmur

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may be audible over the aortic area, and again become apparent along the vertebral column, as low down as the bifurcation of iliac vessels. Such murmurs are also, as a rule, systolic.

The aortic systolic and diastolic murmurs are frequently audible in the *carotids*, and the author has heard a loud diastolic murmur conducted to radial and femoral vessels. Duroziez has pointed out that in aortic regurgitation a double murmur may be heard on light pressure over the *femoral artery*.

Attention must also be directed to the character of the cardiac sounds in the *large arteries in the neck*; thus in severe aortic regurgitation the second sound may be absent in the carotids. Another physical sign of importance is a low-pitched, *musical, second sound* over the arch of the aorta. This points to a general dilatation of the vessel or to an actual aneurysm.

5. ARTERIAL MURMURS

Arterial murmurs are occasionally met with; for example, a systolic murmur in the left subclavian artery may occur in anaemia, and in coarctation or congenital stenosis of the aorta, a systolic murmur may be heard posteriorly close to the vertebral column.

6. VENOUS MURMURS

A venous hum, the *bruit de diable*, is frequently present in the neck, over the internal jugular. It is a continuous, humming sound, rising and falling with the respiratory movements. This can be heard most distinctly by turning the face from the side of the vein that is auscultated. This murmur is often, though not by any means invariably, associated with anaemia.

A similar murmur is sometimes audible in the epigastrium when the inferior vena cava is constricted by a cirrhotic liver.

A SCHEME OF THE VARIOUS CARDIAC MURMURS

Organic Valvular Murmurs

A. *The Cardinal Murmurs.*

(a) *Systolic.*

1. Mitral systolic = mitral regurgitation.
2. Tricuspid systolic = tricuspid „
3. Aortic systolic = aortic stenosis.
4. Pulmonary systolic = pulmonary „

(b) *Presystolic.*

1. Mitral presystolic = mitral stenosis.
2. Tricuspid presystolic = tricuspid „
3. (An unusual murmur, mitral presystolic in character, in some cases of aortic regurgitation.)

(c) *Diastolic Murmurs.*

1. Aortic = aortic regurgitation.
2. Pulmonary = pulmonary „
(very rare).

B. *Combined Murmurs.*

1. Mitral systolic and presystolic = mitral regurgitation and stenosis.
2. Aortic systolic and diastolic = aortic stenosis and regurgitation.
3. Tricuspid systolic and presystolic = tricuspid regurgitation and stenosis.

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4. Pulmonary systolic and diastolic = Some cases of patent
(very rare.) ductus arteriosus,
and where there is
a communication
between the aortic
and pulmonary
artery, the result
of erosion by an
aortic aneurysm.

C. *Murmurs, the result of relative incompetence of valves, always systolic.*

1. Mitral. (a) Secondary to aortic disease.
(b) Primary, due to dilatation of the left
ventricle.
2. Tricuspid. (a) Secondary to aortic and mitral disease.
(b) Primary, due to dilatation of the right
ventricle.

D. *Multiple Organic Murmurs,*

of which the most important are combined aortic and
mitral lesions.

E. *Functional Murmurs, always systolic.*

1. Pulmonary.
2. Tricuspid.
3. Aortic.
4. Mitral.

F. *Cardio-Pulmonary.*

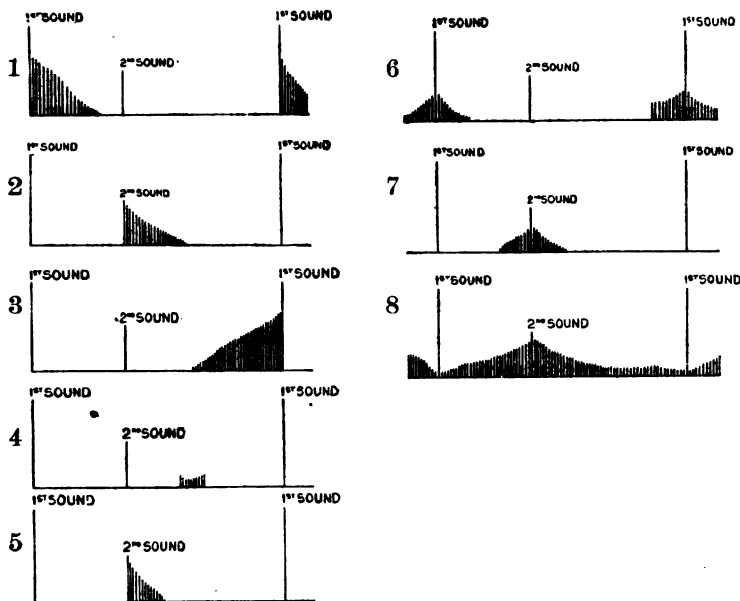


Fig. 3. Diagrams to illustrate the position of the various cardiac murmurs in the cardiac cycle.

1. The systolic murmur—mitral, aortic, tricuspid, or pulmonary.
2. The diastolic murmur—aortic or pulmonary.
3. The presystolic murmur—mitral.
4. The mid-diastolic murmur—mitral.
5. The diastolic murmur—mitral.
6. The to-and-fro murmur—mitral.
7. The to-and-fro murmur—aortic.
8. The continuous murmur in patent ductus arteriosus, or rupture of an aortic aneurysm into the pulmonary artery.

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ADVENTITIOUS SOUNDS

Class II. Friction Sounds

These are (1) Pericardial and (2) Pleuro-pericardial.

1. Pericardial friction is the most important sign of pericarditis.

It is first audible over the *base* of the heart, but may later be heard over the entire precordial area.

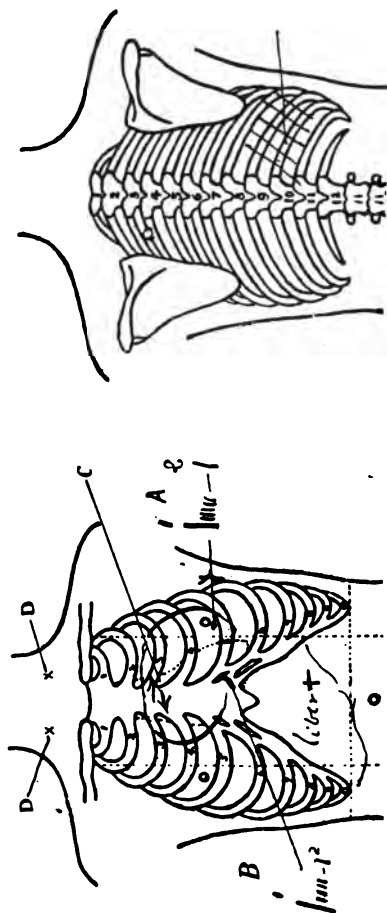
In rhythm it is classically *systolic and diastolic*, but it may be only systolic.

In character, it is rubbing, scraping, or creaking, and in position it is superficial, and can often be modified by light pressure with the stethoscope on the chest, or by deep respiratory movements. These are points of considerable diagnostic importance.

In rare cases, there is, with pericarditis, *air* present in the pericardial cavity. This may occur, for example, when a malignant growth ulcerates from the oesophagus into the pericardium. Splashing, or musical, moist sounds of remarkable intensity, are then audible upon auscultation.

2. *Pleuro-pericardial friction* is caused by inflammation of the adjacent surfaces of the parietal pericardium and pleurae. This sound is scratching or rubbing in character, systolic and diastolic in time, and has a *double rhythm*, an obvious respiratory one, and a fainter cardiac one, which rises and falls with the respiratory movements.

The investigation of a case of cardiac disease is conveniently completed by recording some of the salient facts upon *case book diagrams*. This enables the medical man on a future occasion to refresh his memory of the original condition with great rapidity.



Figs. 4 and 5. Diagrams to show a graphic record of the main features of a case of cardiac disease.

- I. The heart is much dilated, and the liver enlarged. A. Represents a systolic mitral murmur. B. A systolic tricuspid murmur. C. Pericardial friction. D. Indicates enlarged veins. E. Oedema of the lower extremities.
- II. F. Represents a pleural effusion at the right base.

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e. Radiographic Examination of the Heart

This method is essentially a special one, and the interpretation of the results requires much skill and practice.

The fluorescent screen is the most suitable for the purpose.

These landmarks are visible when the normal heart is satisfactorily displayed :—

1. In the first intercostal space on the left side, a part of the aorta.
2. In the second intercostal space on the left, the infundibulum and pulmonary artery.
3. In the second intercostal space on the right, the superior vena cava.
4. The margin of the left ventricle.
5. The right auricle to the right of the sternum, extending from the third to the fifth intercostal spaces. On taking a deep inspiration a light interval appears between the heart and the liver.

Ortho-diagraphy, invented by Moritz of Giessen, enables the skilled operator to estimate the exact size of the heart. Aided by this, the occurrence of cardiac dilatation can be determined, and the results of treatment and exercise tested. Dilatation of the arch of the aorta can be readily discovered, and the diagnosis of aneurysm of this vessel be confirmed, or even made in doubtful cases. A word of warning is needed against the hasty acceptance of the diagnosis of an aneurysm from some apparently unusual pulsating shadow, when there are no clinical symptoms in its support.

I am impressed with the need there is for good technique and careful interpretation in such difficult cases.

Calcified arteries have been detected by this most valuable and promising method of investigation.

CHAPTER IV

THE GENERAL PRINCIPLES OF CLINICAL INVESTIGATION (*continued*)

Div. II. Sect. 1: The pulse—Frequency—Rhythm—Character of wave—Blood-pressure and arterial tension—Estimation of blood-pressure—By the finger—By the sphygmomanometer—Character of the arterial wall—Special points—Unequal pulses—Dicrotism—Anacrotism—Pulsus bisferiens—Pulsus paradoxus—Cardiac hemisystole—*Div. II. Sect. 3:* Examination of other blood-vessels—Arteries—Capillaries—Veins.

Div. III: Secondary damage, the result of the primary heart disease in—Liver—Stomach—Lungs—Kidneys—Spleen—Uterus—Cardiac dropy—Embolism and infarction—Symptoms of embolism in—Lungs—Spleen—Kidneys—Brain—Mesenteric arteries—Abdominal aorta—Peripheral arteries—Thrombosis venous and arterial.

DIV. II. SECT. 1. THE EXAMINATION OF THE PULSE

An examination of the pulse should be careful and complete, and made with three fingers placed upon the radial artery.

The graphic method of studying the pulse by means of the sphygmograph is fully dealt with in books upon physiology.

The use of this instrument in clinical medicine is practically confined to the hospital wards, where it is of much service in registering the characters of the chief variations from the normal, and in impressing them on the memory and furnishing permanent records for future references.

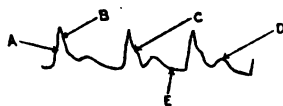


FIG. 6. Normal pulse-tracing.

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The normal pulse-tracing shows an ascending limb (A), apex, and a descending limb (B).

Upon the descending limb there are irregularities representing the tidal wave (C), and the dicrotic wave (D) with its post-dicrotic undulations (E).

A. FREQUENCY OF PULSE

(a) The first point to ascertain is the *frequency* of the pulse, and in difficult cases this should always be checked by auscultation of the heart.

In some conditions as in *severe dilatation* or *advanced mitral disease*, a percentage of the systoles of the left ventricle may be so feeble that no corresponding wave can be detected in the pulse. In such cases examination of the pulse alone would give an entirely wrong impression.

B. RHYTHM OF PULSE

(b) *The rhythm* may be much altered in disease. Extreme irregularity is, for example, a feature of severe mitral disease.



FIG. 7. High pressure. Pulsus bigeminus. From a case of granular kidney with marked arterial disease (T. Lewis).

Irregularity of the pulse without any sign of heart disease may, however, have no pathological significance, and be peculiar to the individual.

Disturbances of rhythm fall into two main groups:—

1. Those in which there is a periodic irregularity, as in

the pulsus alternans where there occurs a large and small beat alternately ; the pulsus bigeminus, trigeminus, &c.

This condition is attributed by Mackenzie to an extra ventricular systole following the regular beat, the result apparently of increase of ventricular excitability.

2. Those in which there is complete arrhythmia.

Gibson emphasizes also the distinction between those forms of irregularity which are met with most commonly in *early life*, and are characterized by varying length of the systole, and the more important forms associated with *organic heart disease*. The former are to be explained as the result of nervous influences *inhibiting* and thus *retarding* the systole, the latter as the result of *premature ineffectual systoles*, the starting-point of the fault resting with the auricles.

C. CHARACTER OF PULSE WAVE

(c) *The character of the wave.* This includes :—

- (1) The duration.
- (2) The size.
- (3) The compressibility.

(1) The duration of the wave is prolonged when the blood-pressure is high and the impulse of the left ventricle powerful, shortened when the reverse is the case.

(2) The extent of the pulse wave is small in some cases of high blood-pressure or when the output from the ventricle is small, as in advanced mitral stenosis. The rise and fall of the wave are abrupt, when the pressure is low, the vessels elastic, and the heart forcible.

(3) *The compressibility of the wave.* This is estimated by gently pressing the three fingers down on the vessel until the wave is obliterated. This investigation necessitates a brief consideration of—

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Blood-pressure and Arterial Tension

One of the chief points to be learnt from investigation of the pulse is information upon the condition of *blood-pressure*. In many treatises we meet with two terms in frequent use, namely, *blood-pressure* and *arterial tension*, both of which must be clearly understood.

The factors in the blood-pressure are, in the first place, the *peripheral resistance* which is a continuous opposing force to the blood-flow, and the *ventricular systole* an intermittent force producing the blood-flow. Between these two forces lie the elastic and muscular arteries.

There are certain other factors concerned in the blood-pressure, and among them are the *viscosity* of the blood, which may be included among the elements of peripheral resistance, and the *volume* of blood discharged by the left ventricle, which is to be ranged with the heart's force.

At the systole of the heart there is an abrupt systolic rise of pressure which the arterial walls indicate by expansion. At the end of systole, the closure of the aortic valve offers an impassable barrier to the return of blood into the heart, and the recoil of the arteries is solely concerned in overcoming the peripheral resistance by the lateral pressure which their walls exert on the volume of blood within them ; this is the *mean blood-pressure*.

If we suppose that these arteries are perfectly *elastic*, then the pressure of blood within them produced by the two opposing forces of the heart and peripheral resistance, would cause a condition of tension in their walls which could be termed the *arterial tension*.

As it is clearly not possible to measure in man the direct blood-pressure, reliance must be placed upon the *lateral blood-pressure*, and if the arteries were perfectly elastic, the

term arterial tension could be used as a measure of this blood-pressure, and in this sense it is often used. Unfortunately the vessel-walls are not perfectly elastic, but are muscular and under nervous control. This at once introduces difficulty, for the arterial tension ceases to be an accurate measure of the lateral blood-pressure, and for this reason I shall entirely discard the use of the former term. When the blood-pressure is under consideration, the term *blood-pressure* will be used, and when the condition of the arterial wall is under consideration, the term *arterial tone*.

The blood-pressure in man is estimated by (i) the *finger* and (ii) by special instruments termed *sphygmomanometers*.

The majority of those who have given particular attention to the clinical estimation of blood-pressure are agreed that the finger is not a reliable gauge, but observers of the greatest experience and the highest reputation are of opinion that very valuable information can be obtained from well-trained fingers both with regard to the blood-pressure and the tone of the arterial wall, and for this reason every effort should be made to cultivate the power of estimating these points.

On the other hand, the *sphygmomanometer* is invaluable for research, for obtaining permanent records, and training the fingers, and experts are agreed that the best types of instrument, if carefully applied, show but a slight margin of error.

(1) In estimating the blood-pressure with the fingers, all three should be placed on the artery, and gentle pressure made until the wave is obliterated, but there must always be a certain tendency to error dependent upon the variation in the *size* of the radial artery, for the larger the artery the higher the pressure appears.

Thus, if we compress first the radial artery and then the

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femoral, the blood-pressure *appears to be higher* in the femoral, in spite of the fact that numerous observations have shown that the pressure does not alter appreciably between the large vessels and minute arterioles. Whenever, then, the radial artery is unusually small and wiry, the tendency of the finger is to *under-estimate* the blood-pressure in the vessel.

(2) The sphygmomanometer is not altogether devoid of error, even in its most modern and improved form. Some of these errors are avoidable with care and practice. Others are unavoidable. When, for example, the arteries are *thickened* by arterio-sclerosis, a certain amount of the pressure exerted by the instrument has to be used up in overcoming the resistance of the artery, and the blood-pressure will be over-estimated. Some of those who have devoted attention to this point are of opinion that this error is an insignificant one, but I am sceptical upon this point.

The reader must be referred to special treatises on the subject for descriptions of the various instruments that have been invented for ascertaining the blood-pressure. Here only one will be described, and its principle and method of application explained.

This instrument is Martin's modification of the Riva-Rocci instrument. It is convenient, the cost is 30s., and it is devoid of serious error.¹

The principle of the method. The pressure is applied by a bag which completely surrounds the limb, and which, for an adult, should be four and a half inches broad. This bag is made of rubber, covered externally by unyielding leather, internally by soft material. When applied, the free ends of the bag must overlap, and they must then be fastened by straps and buckles so that there is a gentle pressure exerted

¹ This instrument can be obtained from T. Hawksley, 357 Oxford Street.

on the limb—as a rule, the arm. When this has been done, the bag is inflated by a rubber ball to which it is

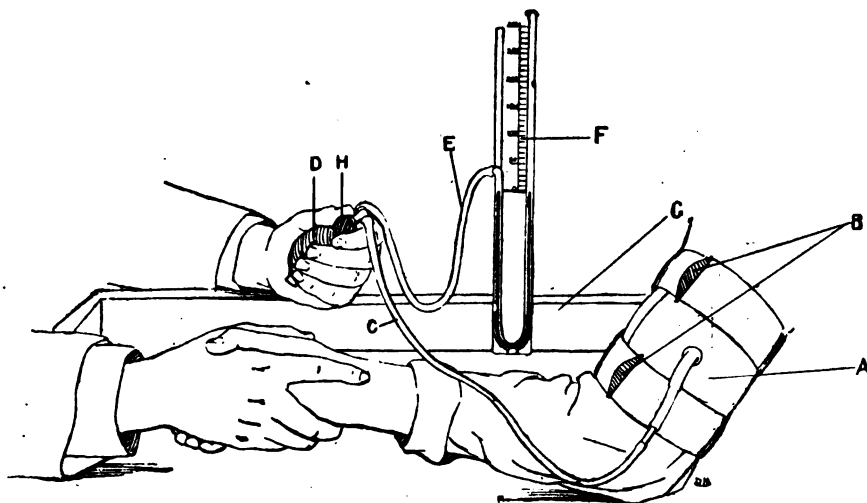


FIG. 8. Martin's modification of the Riva-Rocci sphygmomanometer (Hawksley).

- A. The bag for constricting the limb.
- B. Straps.
- C. Tube leading from rubber ball to bag.
- D. Rubber ball.
- E. Tube leading to manometer.
- F. Manometer.
- G. Stand for manometer.

From the ball there is a short side-tube, closed by a screw (H) and leather washer; the object of this is to allow the escape of the air after the record has been made and before the bag is removed.

Two small rubber caps are provided to close the manometer tubes, so that it can be placed horizontally in its case. Before use these are removed.

The manometer is so graduated that the pressure can be directly read off in millimetres of mercury.

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connected by rubber tubing fitted with a T-piece, the other tube of which passes to a mercury manometer.

When the pressure of the bag has reached a certain limit, the pulse at the wrist will be obliterated, and this pressure is noted on the manometer. The pressure is then gently

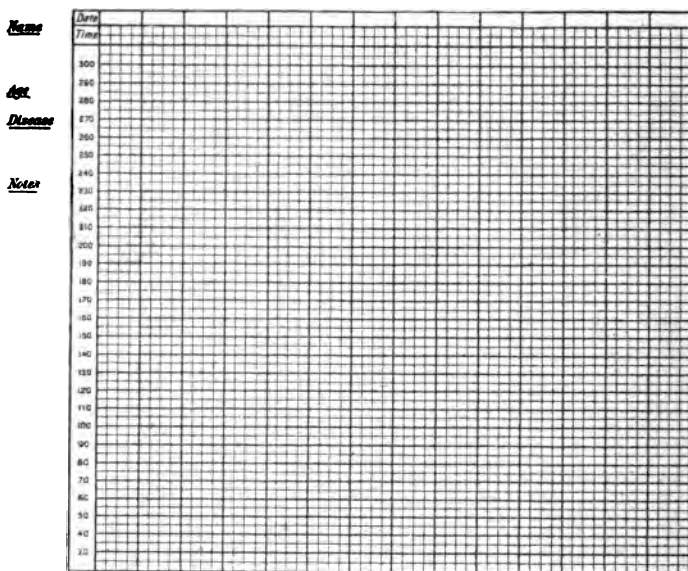


FIG. 9. Chart for recording blood-pressure (H. K. Lewis).

relaxed until the pulse at the wrist reappears, and the manometer reading again taken. This is the *important reading*, and several estimations should be made.

The arm and hand should be well supported at the level of the heart and all excitement avoided.

This method estimates the maximum systolic pressure.

Spasm, tremor, oedema, and obesity interfere with the making of correct estimations.

There are *considerable variations in the normal blood-pressure*—in the young, the systolic pressure may be between 90 and 140 mm. of mercury—the diastolic between 60 and 110.

The normal pressure is highest in the forenoon and rises after mental and physical exertion. It may either fall or rise after meals. The mean pressure is, in particular, raised in the standing position. Lastly, after middle life there would appear to be, as a rule, a steady rise.

Estimations of the blood-pressure can be recorded just as the temperature, upon special charts such as have been devised by Janaway.

D. THE CHARACTER OF THE ARTERIAL WALL

The feel of a normal vessel-wall is elastic, and between the pulse-beats the sensation given to the finger is scarcely distinguishable from that given by the overlying tissues. In arterial diseases the vessel-wall feels *resistant*. This may be the result of a spasm of the muscular coat, or of increase in the thickness of this coat, or again of sclerotic changes in the vessel-wall; or lastly, of calcification. In what is termed, by Clifford Allbutt, the *involutionary or senile* type of arterio-sclerosis, not only are the vessel-walls thickened, but the vessels are tortuous and dislocated from their bed at each pulsation.

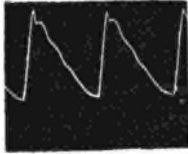
E. SPECIAL POINTS

In some cases of aneurysm there is a marked *difference in the two radial pulses*.

Occasionally the radial artery is abnormal in its distribu-

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tion, and unless this point is remembered needless alarm may be caused by the detection of a difference between the two radial pulses.



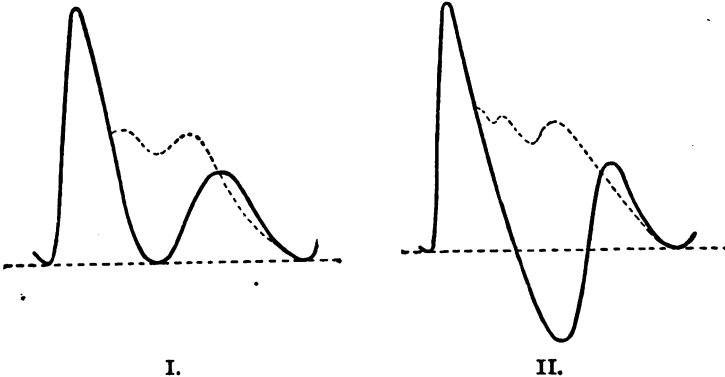
a.



b.

FIG. 10. a. Right radial. b. Left radial. From a case of large thoracic aneurysm. The tracings were taken at equal pressures (T. Lewis).

Dicrotism is a term applied to the pulse when the dicrotic wave is unusually evident. It may in some cases



I.

II.

FIG. 11. Diagrams illustrating (I) the dicrotic, (II) the hyper-dicrotic pulse. (Reproduced from Gibson and Russell's *Physical Diagnosis*, by permission of Messrs. Young and Pentland.)

be so apparent that it is mistaken by the inexperienced for a separate pulse-beat,

The dicrotic wave is looked upon as being produced by the reflection of a pressure-wave from the aortic valve and neighbouring walls of the larger vessels. The chief factors are an *abrupt, short, heart systole* and a *low peripheral pressure*. Dicrotism may be particularly well marked in the later stages of typhoid fever.

Hyperdicrotism is distinguished from dicrotism by the descent of the notch below the base line.

The anacrotic pulse. In tracings of this pulse the *primary elevation* is seen not to reach the highest part of the tracing. Lewis, in a recent article in the 'Practitioner',



FIG. 12. The anacrotic pulse. From a case of aortic stenosis (T. Lewis).

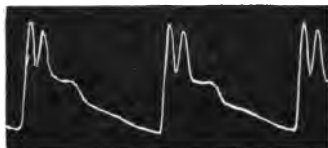


FIG. 13. Pulsus bisferiens. From a case of double aortic disease, with considerable arterial degeneration (T. Lewis).

concludes that it depends upon the loss of the normal relationship between the pressures of the wave entering a vessel and the resistances in the vessel itself. This pulse occurs in aortic disease and aneurysm, and particularly in aortic stenosis.

The pulsus bisferiens. This occurs also in aortic disease, particularly in combined aortic regurgitation and stenosis.

The pulsus paradoxus. As a rule, with inspiration there is an increase in the strength and frequency of the pulse; the pulsus paradoxus shows the reverse. With inspiration it becomes *slower and weaker*, and with expira-

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tion *stronger and more frequent*. Such a condition may occur in good health, but as a general rule such a pulse points to some weakness in the heart's contraction, which permits inspiration to influence unduly the ventricular systole. It may be met with, for example, in general pericardial and mediastinal adhesion.

Cardiac hemisystole. By this is meant contraction of one side of the heart only. In clinical medicine this statement must be modified to the extent that one side of the heart contracts forcibly, and the other so feebly that it cannot be detected. In such a condition there may be a pulse in the internal jugular vein, the result of a right ventricle systole, and not the least sign of a pulse at the wrist. Gibson describes an example of the reverse in which, with a definite left ventricle beat and radial pulse, there was no corresponding ventricular movement on the right side. Cardiac hemisystole points to a most serious condition of heart failure.

DIVISION II SECT. 3. THE EXAMINATION OF THE OTHER BLOOD-VESSELS

a. Arteries. Arterial disease is capricious in its distribution, and on this account accessible vessels other than the radials should be examined. In spite of this precaution, it is impossible to exclude this condition, for *sclerosis of the splanchnic vessels* or *cerebral arteries* may occur when the external arteries might not suggest its presence. Aneurysm of a distal artery may be diagnostic of malignant endocarditis, and sudden arrest of the circulation in an artery points to an embolism. Arterial haemorrhage from the nose in the elderly may prove a valuable warning of *chronic, high blood-pressure* and threatening apoplexy. In the

heart affections of the gouty, and in some cases of angina pectoris and aneurysm, there may be *local arterial spasm*—for example in the fingers—producing local asphyxia.

b. The capillaries are examined for pulsation, which occurs in *aortic regurgitation*, and in some cases of extremely low blood-pressure. Persistent engorgement of these vessels in obstructive heart disease explains the high colour and dusky tinge of the face, and the cyanosis of the extremities. In malignant forms of heart disease, capillary haemorrhages are frequent, and produce *purpuric eruptions*.



FIG. 14. A tracing of the venous pulse. Gibson and Russell's *Physical Diagnosis*. (By permission of Messrs. Young and Pentland.)

c. Veins. The venous pulse in the neck.—There is normally a positive wave in the large veins in the neck which is synchronous with auricular systole, and is termed the *auricular wave*.

Another wave of importance is one which occurs at the end of ventricular systole. This Gottwall and Morrow regard as *always present*, and as produced by more venous blood coming in from the periphery than the auricle can accommodate, with, as a result, venous distension of the large vessels. Morrow terms this the *onflow wave*. Mackenzie and Gibson maintain that it is the result of *tricuspid incom-*

petence. This latter interpretation is of clinical importance, and Mackenzie has traced this short ventricular wave, increasing year by year, until it reaches the obvious venous pulsation which occupies the entire ventricular systole, and is a well-known sign of advanced tricuspid regurgitation.

Venous pulsation in the jugulars must be distinguished from the adjacent carotid pulsation. The distended external jugular should also be emptied by the finger, and notice taken whether it fills from below. Finally, the effect of deep inspiration must be noted, for if this produces no alteration in the distension it points to pressure upon the innominate or superior caval vein by an aneurysm or tumour.

Varicose veins over the chest may be valuable evidence of *aneurysm*; and the direction of flow of the blood in the vessels should be ascertained.

In some cases of general arterio-sclerosis there is also *phlebo-sclerosis*, and in association with active heart disease phlebitis and venous thrombosis may occur.

DIVISION III. THE INVESTIGATION OF SECONDARY DAMAGE, THE RESULT OF THE PRIMARY HEART DISEASE

When compensation fails and there are tricuspid regurgitation and venous stagnation, all the other organs in the body are damaged.

a. The **liver** enlarges, becomes tender, and may actually pulsate. The intralobular blood capillaries are much distended. The liver cells round the central veins are atrophied, and in the middle zone show fatty degeneration. In very severe and chronic cases of tricuspid incompetence there is increase in the connective tissue. The liver on section is found to be mottled brown and yellow, and is termed the *nutmeg liver*.

b. The stomach shows general venous congestion. The mucous membrane is greatly thickened and oedematous, and is coated with tenacious mucus. The muscular tone is feeble and the cavity often much dilated.

The intestines show changes of a comparable nature. These changes help to explain the vomiting and diarrhoea that may occur in advanced heart disease.

c. The lungs. These viscera suffer frequently and severely, situated as they are between the right and left side of the heart, and are often in a state of *chronic congestion*, which particularly affects the bases. This eventually results in an increase of the pulmonary *fibrous tissue* and thickening of the alveoli, with over-distension and often rupture of the blood capillaries, a condition termed *brown induration*. In the final stage, in addition to passive congestion there is often *hypostatic pneumonia*, in which pulmonary oedema takes a prominent part. *Infarctions* and *pulmonary thrombosis* are considered below.

Pleural effusions are frequent and cause pulmonary collapse, thus adding to the dyspnoea.

d. The kidneys are large, firm and cyanotic. Post mortem, the capsules strip with undue ease, and show a swollen, dark purple cortex full of venous blood. The urine is scanty, high-coloured, loaded with urates, and often albuminous. In addition, *infarction* and *nephritis* may complicate heart disease.

e. The spleen is generally small, though sometimes increased in size, and unless there is infarction or chronic septicaemia, usually firmer than normal.

f. Congestion of the uterine mucous membrane may produce metrorrhagia or menorrhagia.

g. The altered cerebral circulation produces mental deterioration, and both cerebral thrombosis and embolism are

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complications which are met with in heart disease as a direct outcome of the cardiac affection.

Cardiac dropsy. This commences as a rule in the *most dependent parts*, and is particularly liable to supervene in mitral lesions. The earliest symptom is some puffiness around the ankles noticed at the end of the day. Dropsy is a much more prominent feature in adult heart disease than in childhood. The serous membranes may be affected, and there may be great ascites, especially in mitral stenosis. The explanation of cardiac dropsy is difficult and the causation complex.

Starling, in 1896, emphasized the importance of hydraemic plethora, and held that as a result of the failure of compensation there was a fall in the arterial and capillary blood-pressure, and a rise in the venous. This disturbed the normal relation between absorption and filtration through the capillaries, with the result that excess of fluid was absorbed and *hydraemic plethora* resulted. As a result of this plethora, combined with damage to the capillary wall from venous blood, there then resulted an increased filtration from the vessels into the tissues and a consequent dropsy. Bolton has, however, produced dropsy in animals by constriction of the heart. This was effected by gathering up the pericardium around it by ligation. Then followed a brief fall in the arterial and a rise in the venous blood-pressure; but there was speedy adjustment, and within a few days these pressures were again practically normal. Hydraemic plethora would thus appear to be of no vital importance, and Bolton strongly inclines to the view that the most important factor in cardiac dropsy is an alteration in the *permeability of the capillary wall*. Vidal and Lemièrè have shown that in some cases of renal dropsy there is a retention of *chlorides* in the tissues, the result of the renal insufficiency. Further,

under normal conditions, the blood and tissues are isotonic as regards sodium chloride at the level of 0.9 per cent.

When, however, there is retention of chlorides, this isotonic level is raised, and water, as a result, retained in the tissues until they and the blood are once more on a level. This may then be a factor in some cases of cardiac dropsy in which there is renal inadequacy as toward the chlorides. Other factors concerned in cardiac dropsy are the poor *tone of the connective tissues and muscles*, and the circulation of *poisons* in the blood, as exemplified by the extreme and hard oedema in some drunkards with dilated hearts.

In childhood, oedema of the face is sometimes seen resembling closely a renal dropsy, and yet with the urine showing no albumen or other sign of nephritis.

Embolism. This, in heart disease, is the blocking of a vessel by a blood-clot or fragment of vegetation, carried to the vessel by the arterial circulation.

An embolism may result in heart disease from

- (1) A thrombus detached from the heart or from a vein.
- (2) From a fragment of vegetation torn from a damaged valve.

In character it may be (1) simple, or (2) infective.

Pulmonary emboli have their origin in the systemic veins, the right side of the heart, or pulmonary artery. *Systemic emboli* are derived from the pulmonary veins, left side of the heart, or some more proximal artery.

The order of frequency in which embolic manifestations have been detected is, according to Walshe : (1) Pulmonary. (2) Renal. (3) Splenic. (4) Cerebral. (5) Vessels of lower extremities. (6) Of upper extremities. (7) Coeliac axis. (8) Retinal. (9) Mesenteric. (10) Coronary arteries.

When an embolus is impacted in a peripheral vessel, two results may follow ; the first a *mechanical one*, due to the

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obstruction of the circulation, the other, only met with in the infective emboli, *local changes*, the result of the infection.

1. **Results due to obstruction of the circulation.** Collateral circulation is as a rule very shortly established, but necrosis of the area supplied by the obstructed vessel results. The area is usually wedge-shaped, unless it is of large size, and is termed an *infarction*.

The colour of the necrotic area is either *white*, or *red* from haemorrhage, and these infarcts are called respectively white and red. The white infarct is *not a late stage* of the red, but the result of a necrosis without the addition of haemorrhage.

The source of the blood in the haemorrhagic infarct is apparently not a reflux from the veins leaving the damaged area, but a result of ruptured vessels in the capillary anastomoses which take place in the damaged area.

The simple embolus is in course of time replaced by a gradual invasion of *connective tissue elements*.

2. **When the emboli are infective.** The infective agent need not necessarily get a fresh foothold at the seat of infarction, but should it do so it will produce *local changes* depending upon its nature. The most important of these are *inflammation*, *suppuration*, *gangrene*, and *toxaemic haemorrhage*. *Aneurysm* of the affected artery is also a consequence of this condition if a vessel of considerable size is plugged.

SYMPTOMATOLOGY

Only the symptoms of the most important infarctions are considered, and these are described for the sake of brevity in a somewhat arbitrary manner.

I. **Pulmonary embolism.** (a) If a large vessel is occluded, rapid death follows with these symptoms:—

- (1) Sudden precordial pain and anguish.

- (2) Dyspnoea.
- (3) Cyanosis, followed by pallor.
- (4) Rapid and irregular action of the heart with failure of the pulse.
- (5) Restlessness, convulsions, and fatal syncope.

Cardiac murmurs have been detected, but the condition is so distressing that accurate investigation is usually impossible. Great importance must be attached to the combination of *intense dyspnoea with a good air entry*.

b. When the occlusion is partial, or smaller vessels are affected, the symptoms may last for some days, and recovery may occur.

The symptoms of *pulmonary infarction* that is, embolism of medium-sized or small vessels, are:—

- (1) Sudden pain in the chest.
- (2) Fever.
- (3) Cough, haemoptysis, and dyspnoea.
- (4) Pleural friction over the area of infarction.
- (5) Signs of consolidated lung in the same position.

In many cases pain and haemoptysis are the only two indications.

c. *Embolism of small vessels in the lungs* may give rise to no symptoms, collateral circulation being rapidly established.

II. The symptoms of *splenic infarction* are:—

- (1) Sudden pain in the lower part of the chest on the left side.
- (2) Enlargement and tenderness of the spleen.
- (3) In some cases a friction sound due to the inflammation of the splenic capsule over the base of the infarct.
- (4) A sudden rise of temperature.

This infarction may be latent.

In some cases of malignant endocarditis there may be very great enlargement of spleen without the occurrence

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of any infarction. This is apparently a result of the general toxaemia.

III. The symptoms of renal infarction are :—

(1) Sudden pain in the region of the kidney, and tenderness on pressure over the organ.

(2) Sudden haematuria, with the presence of blood and sometimes casts in the urine. The haematuria may be profuse.

(3) A rise of temperature coincident with the infarction.

Although these are the classical symptoms, infarction may occur without any sudden pain or rise of temperature, and be indicated only by haematuria.

In malignant endocarditis again, acute nephritis may develop without infarction. When this is the case there are blood, albumen, and casts present in the urine. The casts are, however, usually more numerous than in infarction; the haematuria is less, and often makes the urine only smoky in colour, the albumen is greater in quantity, and lastly, there may be evident signs of acute renal disease.

IV. Cerebral infarction. The symptoms will depend upon the localization. The most frequent result is a *sudden hemiplegia*, with or without a loss of consciousness.

V. Mesenteric infarction. The usual results are :—

(1) Sudden abdominal pain ;

(2) Vomiting ;

(3) Melaena ;

(4) Meteorism ;

(5) Collapse.

VI. Of the Abdominal aorta :—

(1) Sudden paraplegia, with abdominal pain ;

(2) Obliteration of the pulses in the femoral arteries.

VII. Of the peripheral arteries :—

(1) Sudden pain in the limb ;

- (2) With blanching of the distal parts ; and
- (8) Obliteration of the pulse in any branch beyond the seat of lesion.

Thrombosis, both venous and arterial, may be associated with active morbus cordis or with chronic cardiac incompetency. On six occasions I have seen venous thrombosis occur in the neck or upper extremities associated with acute or subacute rheumatic heart disease.

Hamilton maintains, and many agree with his view, that pulmonary infarctions in chronic cardiac inadequacy are frequently not embolic in origin, but the result of a *gradual arterial thrombosis*.

Thrombosis may also result from diseases of the arteries and veins themselves, and in some cases of great cardiac weakness ante-mortem thrombosis occurs in the heart ; the favourite sites are the auricular appendices and ventricular apices, and the usual condition is one of extreme dilatation of the cavities.

CHAPTER V

THE PATHOLOGY OF RHEUMATIC HEART DISEASE

Acute and subacute lesions—Classification of—Pericarditis—Simple—Malignant—Chronic sclerosing—Endocarditis—Simple—Malignant—Chronic sclerosing—Myocarditis—Acute—Subacute.

In this and the following chapters, the intention is to give as complete a picture as possible of the various cardiac lesions that may rise from a single cause. I think that in a small treatise such as this, a considerable advantage is thus gained; for the pathology of heart disease is made clearer, a more connected view is obtained of the clinical history of cardiac disease, and the indications for treatment become more apparent. It is again easier to realize by this method of description the important distinction between symptoms which are the result of the cause producing the heart disease, and those which are the direct outcome of the damage to the heart. Lastly, other forms of morbus cordis are seen in a more correct perspective.

Rheumatism has been chosen as the type because it is the most frequent cause of heart disease; it attacks young and old, damages all parts of the cardio-vascular system, may end fatally or be recovered from, and finally, may be acute, subacute, or chronic in its course.

ACUTE AND SUBACUTE RHEUMATIC HEART DISEASE

Classification of the Lesions

In acute and severe rheumatic heart disease all parts of the heart are affected, and the condition which results is called

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carditis. It is, however, for clinical purposes, essential to study the rheumatic affections of each part of the heart in detail, for in some cases the valves are the seat of the chief damage, in others the pericardium is extensively implicated, and in others, again, the muscular wall.

These various, active, rheumatic lesions may be thus tabulated:—

- I. { 1. Acute and Subacute Pericarditis. { *a*. Simple.
 { 2. Chronic sclerosing pericarditis. { *b*. Malignant.
- II. { 1. Acute and subacute endocarditis. { *a*. Simple.
 { 2. Chronic sclerosing endocarditis. { *b*. Malignant.
- III. Acute and subacute myocarditis.
- IV. Multiple serositis.

THE PATHOLOGY OF RHEUMATIC HEART DISEASE

This form of heart disease is the type of a cardiac infection.

The infective agent is the *diplococcus rheumaticus*, a minute diplococcus belonging to the large group of streptococci, and closely allied in its characters to the pneumococcus upon one side and the pyogenic streptococci on the other.

This micrococcus makes its way into the system from the *tonsils* which are frequently found either actively inflamed or unhealthy in acute rheumatism. There are probably *other sites* of infection, but this one only has been conclusively demonstrated.

In fatal carditis the diplococcus has been demonstrated in the damaged valves, in the pericardium, pericardial exudation and myocardial connective tissue; it produces carditis in animals, and has been demonstrated in these lesions also.

Many bacteriologists do not accept this statement as to the cause of rheumatic carditis, but look upon the infection

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as still unknown. The future alone can settle this point, but of their objections to the diplococcus rheumaticus this can be stated, that they are chiefly negative.

Should, however, the reader be unable to accept the diplococcus, the facts that are stated here about the morbid anatomy will still hold good, and he will only need to apply some other interpretation—if such exists—in explanation of their occurrence.

I. PERICARDITIS

(a) Simple

The first step in pericarditis is the deposition of the micro-organisms from the capillaries of the coronary vessels in the *subendothelial tissue* of the visceral and parietal layers of the pericardium. There result hyperaemia and capillary haemorrhages. The connective tissue in the neighbourhood swells, and in severe cases undergoes coagulation necrosis, and there is also an exudation from the capillaries of serum, and a diapoechesis of leucocytes. In this way swelling and weakening of the pericardial tissues are produced.

The *endothelium* is soon involved where it overlies the foci of disease, the cells are destroyed, and from the free surface of the membrane there is an exudation of fluid which differs in character with the severity of the process. If this is very acute, the effusion is haemorrhagic, if less so it is at first serous, then sero-fibrinous, and lastly, if persistent and acute, sero-purulent.

It is often stated that rheumatic lesions do not suppurate, a dictum I cannot accept, for it has been my experience to meet with cases of pericarditis in which the condition of the effusion was certainly one of rheumatic suppuration. The micrococci are located at first beneath the endothelium, but when this is extensively damaged they will almost invari-

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ably be found in films of the exudation, if these are deeply stained with methylene blue, and examined under $\frac{1}{2}$ th objective.

The *effusion* in rheumatic pericarditis is seldom large in amount, as it is in some cases of renal disease and 'suppurative' pericarditis. Thus in 150 cases of fatal rheumatic heart disease in childhood, in only one were six ounces of fluid found in the pericardium.

The *reparative processes* in pericarditis consist in the destruction of the micrococci by the leucocytes, connective tissue, and endothelial cells; and in a repair of the necrotic tissue by scar formation. The fluid that has been exuded is in the meantime absorbed, and layers of fibrinous deposit are slowly organized.

When the endothelium has been greatly damaged, the opposed surfaces of the pericardium come in contact and eventually become *adherent* to one another. These adhesions are at first soft and vascular, and later become firm, and as the infection of the pericardium is produced by numerous different foci, it results that the pericardial adhesions may be general or, as is more often the case, partial.

(b) Malignant Pericarditis

Malignant pericarditis is a rare condition characterized by the severity of the process and the failure of the tissue reaction to cope with the infection. These are shown by the very great thickening of the pericardium, the occurrence of a haemorrhagic exudation, and in somewhat less severe cases by extensive deposit of fibrinous masses upon the surfaces of the membrane.

As is the case with endocarditis there is no sharp line to be drawn between the simple and malignant forms, for there are *border-line cases* in which imperfect recovery occurs, and the virulence of the attack is borne witness to by very

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dense and thick adhesions, in which may be found nodular masses of necrotic tissue.

(c) Chronic Sclerosing Pericarditis

Chronic sclerosing pericarditis is a more frequent occurrence than the preceding.

In this case, although reparative processes are occurring in some parts, the disease remains active in other parts and *smoulders obstinately on* for weeks and months.

It is, in fact, as difficult to be sure that the rheumatic infection has been destroyed in the tissues as it is to feel confident of the arrest of a tuberculosis.

The outcome of this process is the formation of dense adhesions. The whole pericardium may be infiltrated by the disease, and the surrounding mediastinal tissues and adjacent pleurae may also become involved.

In this way also, if other serous surfaces are affected, may originate one of the forms of the condition termed *multiple serositis*.

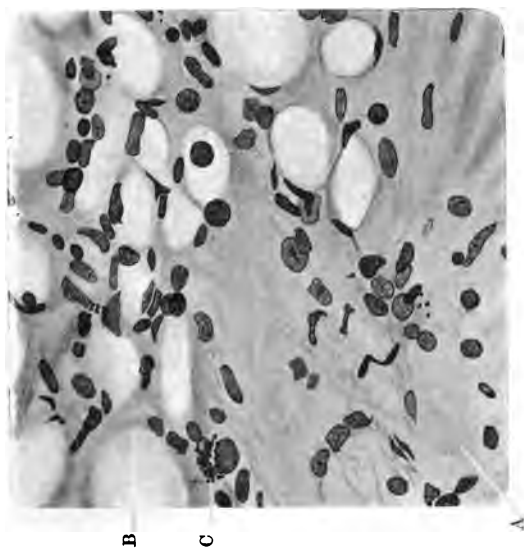
II. ENDOCARDITIS

Acute and Subacute Endocarditis

(a) Simple

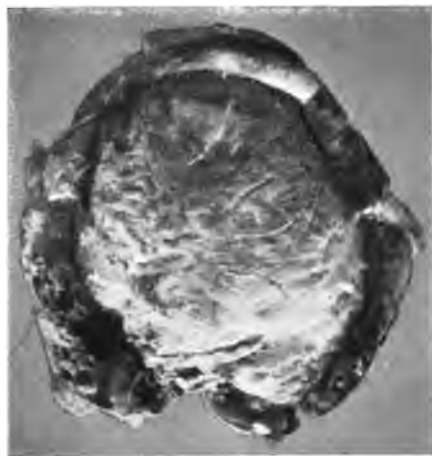
The bacterial infection is carried by the blood capillaries to the *base of the valve*, and thence to the structure of the valve itself by means of the minute channels, by which nutritive material is supplied to the cusps. The bacteria locate themselves in the *subendothelial tissue* at the margins of the cusps, and produce foci of swelling and necrosis. As a result the endothelium of the margin of the valve is raised in a series of small projections, and in many places is destroyed. The necrotic and swollen tissue of the valve projects from the general surface and forms a series of small

PLATE IV



A
Experimental pericarditis (rabbit), produced by intravenous inoculation of the diplococcus rheumaticus, showing A, myocardium; B, visceral pericardium; C, diplococci. (Poynton and Paine.)
By permission of the Pathological Society.

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B
Rheumatic pericarditis (man). The pericardium is reflected, and the shaggy surface of the visceral pericardium is shown.
By permission of Messrs. Lea Brothers, from Carr's 'Practice of Paediatrics'.

PLATE V



A

Simple rheumatic endocarditis (man). The mitral valve is exposed, and shows a crop of vegetations along the edge on the auricular surface.

By permission of Messrs Lippincott.



B

Simple rheumatic endocarditis (rabbit), produced by intravenous inoculation. (Poynton and Paine.)

By permission of Messrs Lippincott.

Between Plates IV and VI]

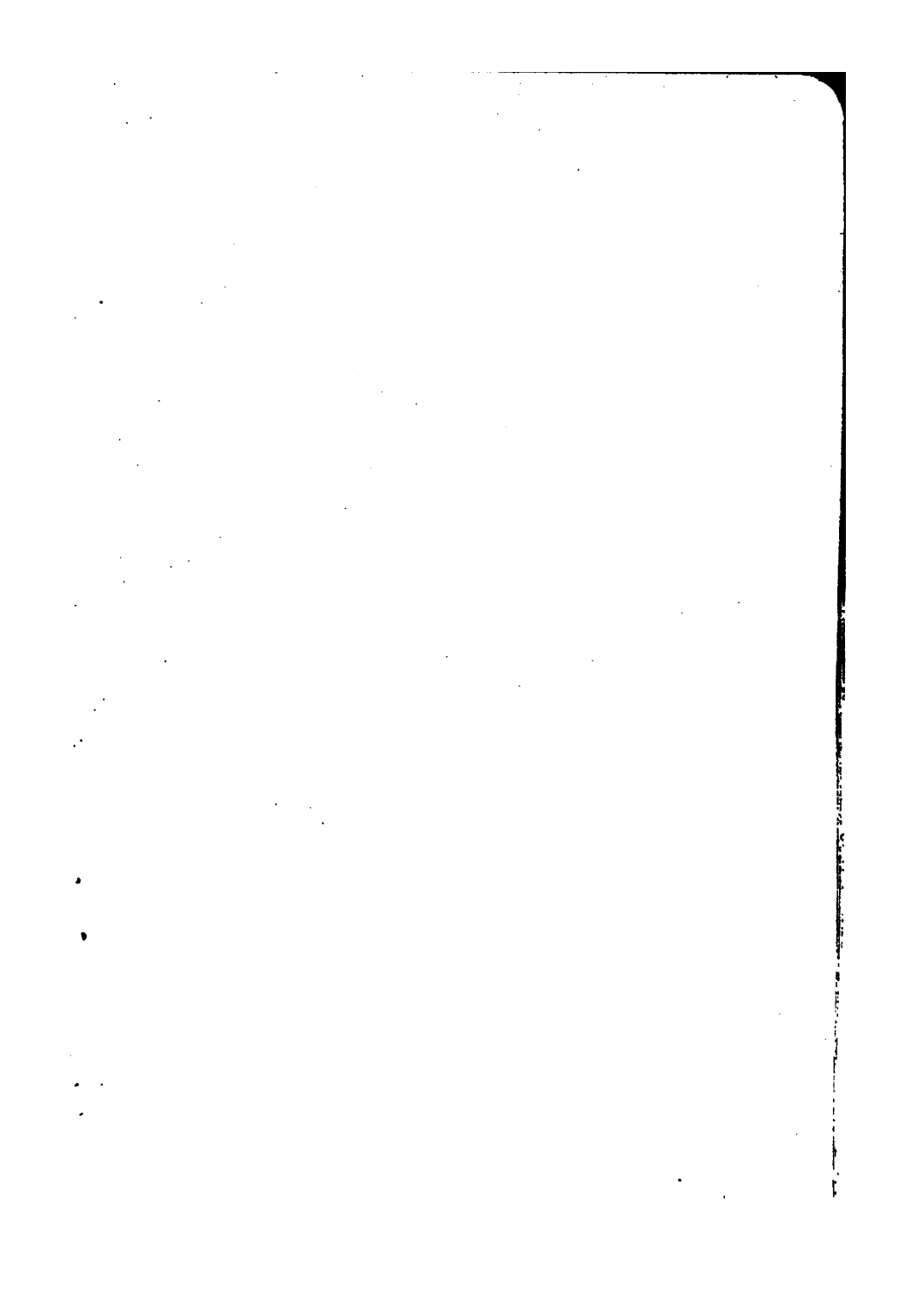
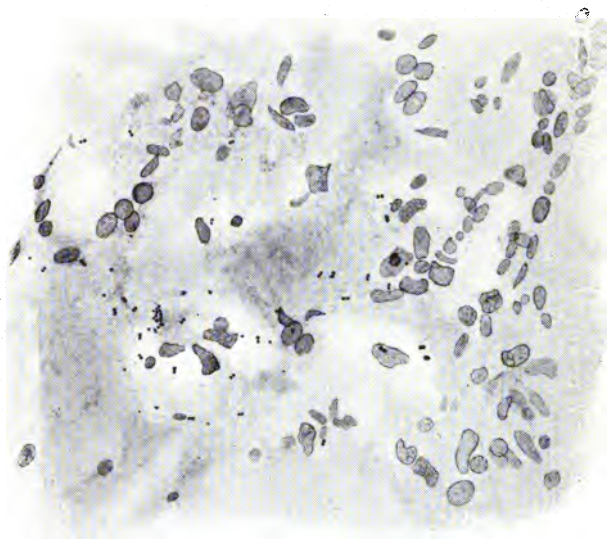


PLATE VI

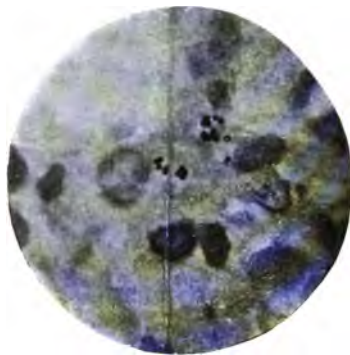


A

A section through a simple vegetation, to show diplococci present in scanty numbers. (Foynton and Paine.)

By permission of 'The Lancet'.

To face p. 86]



B

A micro-photograph showing the diplococci in experimental simple rheumatic endocarditis. (Forbes-Tulloch.)

By permission of 'The Lancet'.

vegetations, which in an early stage are red in colour, and later, yellowish-white. They are firmly attached to the substance of the valve, and consist microscopically of necrotic connective tissue and endothelium, infiltrated with leucocytes, and they merge gradually into the general structure of the valve. Upon them fibrin may be deposited from blood in the heart, but this fibrin is *not an essential constituent* of a vegetation. The healing process consists in the gradual invasion of the vegetation from its base by fibroblasts, and the subsequent formation of fibrous tissue which produces some amount of puckering and thickening of the damaged cusps.

Simple endocarditis is not a fatal condition, and in post-mortem examinations upon cases of rheumatic carditis, the endocarditis which is almost invariably present is, as a rule, in the early reparative stage, the immediate cause of death having been peri- and myocarditis. Sections of the vegetations show that the bacteria are *all or nearly all destroyed*, or are dying and stain feebly. This point will be again alluded to when the pathology of malignant rheumatic endocarditis is considered.

Relative incidence of Endocarditis upon the various valves. The different valves are damaged with varying degrees of frequency.

By far the most frequently attacked is the *mitral*. Next in order come the *mitral and aortic valves* together. Then the *aortic valve* alone. Less frequently the *tricuspid*, and lastly, the *pulmonary*.

When the tricuspid valve is damaged the mitral is also, and not infrequently the aortic as well.

It is in foetal rheumatic endocarditis that the pulmonary valve suffers. Any damage to it in acquired rheumatism is so rare as to be a pathological curiosity.

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(b) Acute and { Malignant or } Endocarditis Subacute { Pernicious }

In this condition the valves are often *ulcerated* or large *fungating vegetations* are found upon them.

It has long been known that the most frequent antecedent in the history of this condition is an attack of *rheumatic heart disease*, although this is not an invariable occurrence. Another recognized fact is, that after death from malignant endocarditis the vegetations are found to contain numerous *micrococci*, whereas in simple acute rheumatic endocarditis they can only exceptionally be detected.

The natural explanation of these two facts used to be, and according to some authorities still is, that a secondary infection had attacked a valve already damaged by previous rheumatism. I have no doubt that in a certain percentage of these cases this is true, but it is *not the whole truth*, and experimental investigation supplies another explanation. When it had been demonstrated by experiment on animals and by observation in man, that simple rheumatic endocarditis was in reality a bacterial infection of the valves, there at once arose the suspicion that this infection might also produce the malignant type of endocarditis, and that some cases of malignant endocarditis might be in reality *truly rheumatic*. Clinical observers had in fact pointed out this probability before any experimental investigation was at hand to support the assertion. We now know that there is strong evidence in its support. Paine and the writer were able in 1902 to supply these links in the chain of evidence, by showing, firstly, that the diplococcus, which is a cause of rheumatic fever, can be isolated in *pure culture* from the vegetations in some cases of malignant endocarditis occurring in rheumatic subjects. Secondly, that this diplococcus, when isolated from simple endocarditis in man, can produce *both simple and malignant*

PLATE VII



A

Malignant endocarditis (rheumatic) of the aortic valve (man).
By permission of Messrs. Lippincott.



B

Section through the necrotic tissue of the vegetations, showing masses of diplococci (man).
By permission of the Royal Medico-Chirurgical Society.

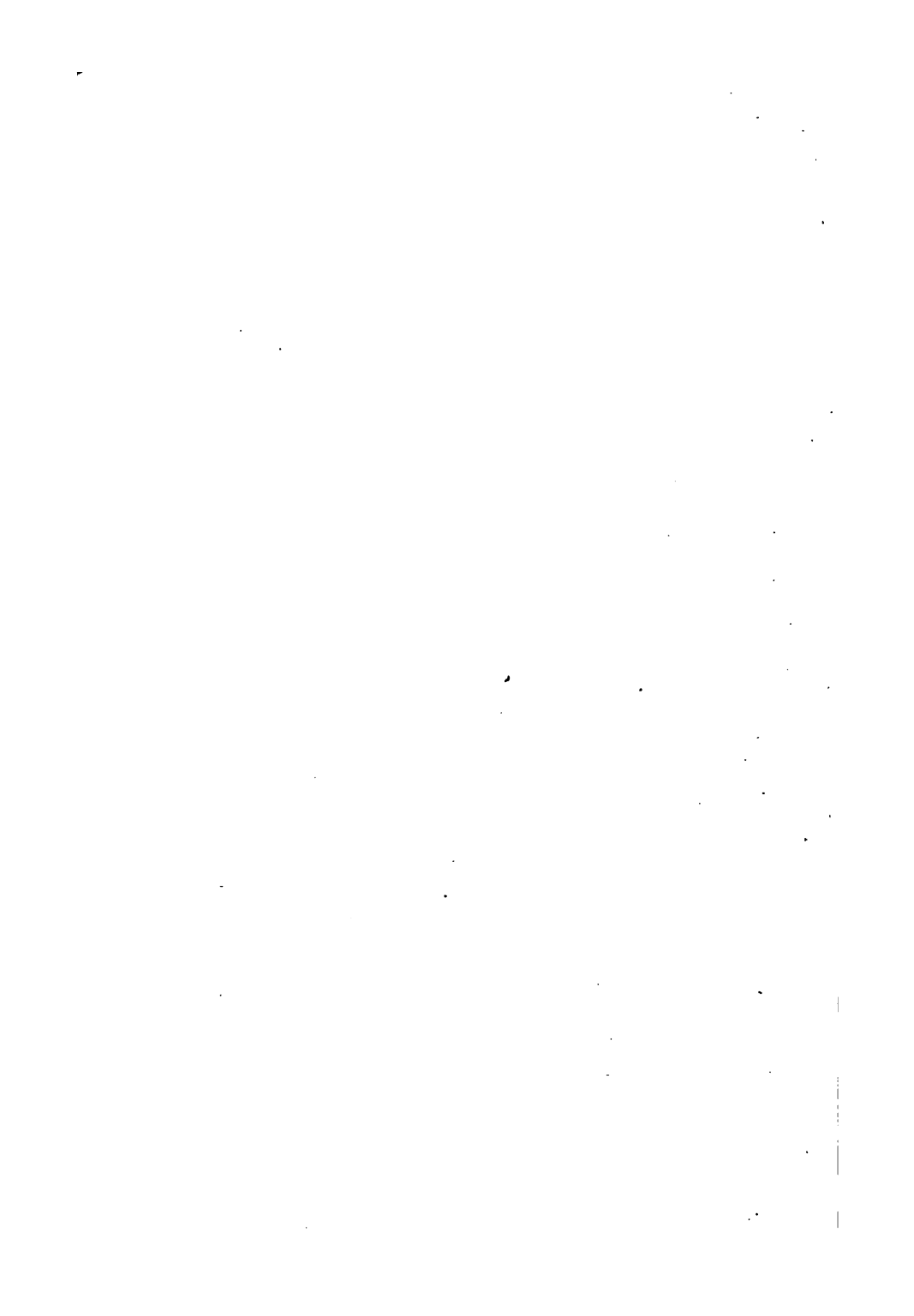


PLATE VIII



A

Experimental rheumatic malignant endocarditis, mitral valve (rabbit). The left auricle and ventricle are opened, showing the endocarditis.
By permission of the Royal Medico-Chirurgical Society.



B

Section through the necrotic tissue of the vegetation, showing masses of diplococci (rabbit).
By permission of Messrs. Lippincott.

endocarditis in animals, and, when isolated from malignant endocarditis, can also produce the same results. Lastly, in man *every gradation* can be traced between the two conditions, both as regards their morbid anatomy and clinical symptoms, and in animals also there exists every gradation between the two conditions.

The present position of malignant endocarditis would thus be that every infection which attacks the endocardium may produce a malignant endocarditis, and the rheumatic infection must therefore be included.

These cases of malignant rheumatic endocarditis are characterized by the absence of ordinary suppuration. Even when an aneurysm results from the impaction in an artery of an embolus carrying the rheumatic infection, such an excellent nidus as blood-clot does not produce suppuration. The formation of white infarcts, on the other hand, is a frequent occurrence. Again, the course is often a protracted one, and the subacute form occurs more frequently in rheumatism than in other infections. And lastly, it is probable that a certain number, after showing all the symptoms of malignancy, gradually quiet down, leaving eventually traces of their former virulence in the shape of bulky scars upon the valves.

The malignancy of the endocarditis does not depend upon the size of the vegetations, or upon the extent of the lesions, or even upon the degree of ulceration. The tests of the malignancy are the *number and virulence of the micrococci* in the damaged valve.

The most malignant cases are those in which there are small vegetations (no larger, it may be, than those met with in simple endocarditis) yet vast numbers of diplococci.

Somewhat less malignant are those in which there are large fungating vegetations and numerous diplococci.

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And the least malignant are those in which there are large vegetations, which are firm in consistence and in which the diplococci are not extremely numerous.

Large vegetations usually mean an attempt at resistance, although such vegetations may sometimes form with great rapidity, and they then are but a very inefficient barrier.

In the firmer vegetations great numbers of bacteria are destroyed, and attempts at scarring may be sometimes detected.

The processes in the formation of the vegetations are comparable to those already described in the simple form, but the necrotic tissue is full of virulent bacteria, and upon the vegetations fibrin is very frequently deposited from the blood in the heart, and this in turn becomes infected. The disease is not so localized as the simple endocarditis, but may spread on to the surface of the auricles and ventricles. The ulcerative process, again, may destroy the chordae tendineae or make a hole in the cusp of a valve. The wall of the heart in the region of the damaged valve may be eroded, and a cardiac aneurysm or rupture of the heart result.

Lastly, when large and friable vegetations teeming with bacteria project into the blood stream, fragments are torn off and driven in every direction by the circulation, thus producing that most dangerous secondary complication termed *septic infarction*.

(c) Chronic Sclerosing Endocarditis

In this form there is a persistent and relapsing infection which, as a rule, *never reaches a high grade of virulence*, although in some cases the character may alter and the malignant type supervene.

It is practically impossible to ascertain when this process is arrested, although there is good evidence to show that such

may occur. It is almost equally difficult to date its commencement. The damage produced is most serious, for it is in this way that the *slow contraction* of a valve is produced. In some cases there is a uniform sclerosis of the ring and the segments of the valve, resulting in the production of a small circular opening which may only admit the top of a pencil. If in addition the chordae tendineae are similarly affected, the segments are tethered down as well as welded together, and the aperture becomes funnel-shaped.

Such complex results as these are only seen in the auriculo-ventricular valves; in the sigmoid valves, the delicate cusps are only thickened and fused together, although in this way an extreme stenosis may result.

This insidious process is much more common in the *female* than in the male sex, and I believe it represents a general law in connexion with the rheumatic infection, to the effect that rheumatism is more chronic and less acutely fatal in females, a law applicable not only to the cardiac lesions, but to the arthritic and nervous ones also. It is unlikely that such a condition as mitral stenosis is produced by the healing of an ordinary acute simple endocarditis, for if this were so, this condition would be constantly met with in childhood. A great many children under eight years of age suffer from endocarditis, yet at twelve years mitral stenosis is rare, although more than ample time has elapsed for the healing of an endocarditis which occurred at the age of eight.

The more carefully the history of mitral stenosis is investigated, the more is one convinced that it is a special type, the result of a *smouldering infection*, lasting over many years; and it is interesting to notice how frequently it is associated with another smouldering manifestation of rheumatism, namely, relapsing chorea.

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III. MYOCARDITIS

(a) Acute

For want of a better term, this one of myocarditis has been retained ; it is, however, not really satisfactory, because the changes in the myocardium are not wholly inflammatory, but the result of a toxic process due to rheumatic infection.

Uncomplicated *acute, fatal cases* of myocarditis are very rare, for there is almost invariably pericarditis as well.

The same is true of experimental infections, the great majority of the animals in whom the heart is affected die from pericarditis and endocarditis, but occasionally myocardial failure only is discovered after death.

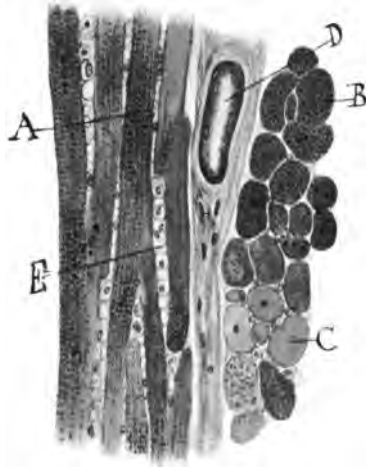
The morbid changes are of two types :—

1. Those affecting the muscle.
2. Those affecting the blood vessels of the heart and their surrounding connective tissue.

1. The most definite change in the muscle is *fatty degeneration*. This occurs in scattered patches throughout the thickness of the myocardium, and particularly in the neighbourhood of the small blood vessels. Such changes are also met with in many cases of rheumatic pericarditis, but that they are not the direct result of that lesion is clear from the fact that they may occur independently. In the fatal cases complicated with pericarditis the fatty changes in the cardiac muscle are particularly well marked beneath its visceral layer. This is explained by the great vascularity of the visceral pericardium, and the greater likelihood of the deposition there of the diplococci.

Other morbid processes are also met with in the muscle in acute myocarditis, such, for example, as chromatolytic changes in the nuclei, loss of striation, and fragmentation of the muscular fibres. Aschoff has laid stress on the possi-

PLATE IX



A section of the wall of the left ventricle from a case of acute rheumatic carditis, showing fatty change in the muscle.

A = Muscular fibre in longitudinal section.

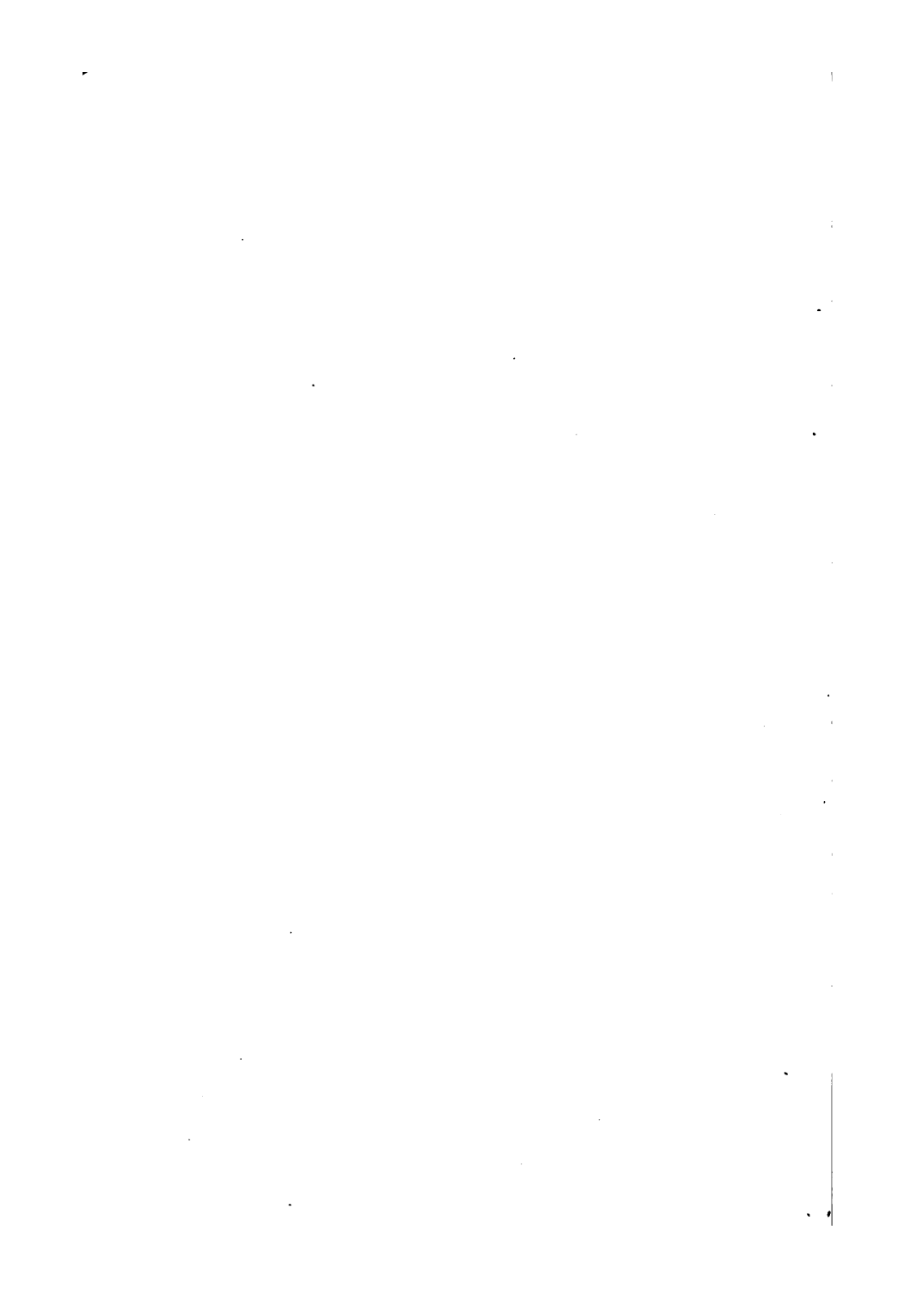
B = " " transverse section.

C = A healthy fibre in transverse section.

D = A blood-vessel

E = Blood capillary between the muscular fibres.

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bility that in some cases the auriculo-ventricular bundle of His is singled out for particular damage.

2. *The changes in connexion with the blood vessels* are congestion, and rupture of capillaries, deposition of diplococci in the perivascular spaces, and the exudation of leucocytes both mono-nuclear and polymorpho-nuclear. The connective tissue supporting the vessels and separating the muscle bundles is the seat of exudation, and becomes swollen, hyaline in appearance, and sometimes necrotic, and Aschoff has described a nodular affection of the myocardium. Such conditions as these are very extreme ones, and rare. When, however, we turn to the clinical side of rheumatic heart disease we find that the most constant evidence of cardiac rheumatism is *acute dilatation of the heart*.

The dilatation is evidence of loss in the contractile power of the myocardium, but it is not necessarily fatal, and complete recovery may frequently occur. It must be presumed in this instance that there is some toxic effect upon the myocardium, but it is very doubtful whether such a change would be demonstrable by ordinary histological methods.

(b) Subacute and Chronic Myocarditis

In this condition there is increase in the connective tissue framework of the heart, the result of the previous acute inflammation. In rheumatism this fibrosis of the heart seldom reaches any considerable degree, and at the present time it is difficult to write with any confidence of a chronic sclerosing myocarditis of rheumatic origin.

Turning again for a moment to the clinical side of the subject, just as one recognizes cases of acute dilatation, so also there is a group in which, without valvular damage,

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there remains a *dilatation and irritable weakness* of the myocardium. One surmises some damage to the muscular fibres, which they are slow to cast off, and compares them with lingering cases of post-diphtheritic and post-influenzal cardiac debility. Such cases as a rule eventually recover, or are later complicated by other rheumatic lesions of the heart, and so the exact proof of this surmise is difficult to obtain.

CHAPTER VI

SYMPTOMATOLOGY OF ACTIVE RHEUMATIC HEART DISEASE.

Acute pericarditis—Simple—Malignant—Chronic sclerosing—
Diagnosis of pericarditis—Endocarditis—Simple—Of Mitral—Aortic—
Tricuspid valves—Malignant—Chronic sclerosing—Mitral—Aortic—
Tricuspid—Myocarditis—Simple—Malignant—Multiple rheumatic
serositis.

THIS chapter is devoted to the most important of the symptoms and physical signs that are met with in active rheumatic heart disease.

It must be repeated that in many instances we are dealing with a rheumatic carditis, but the plan adopted here will be to follow the general scheme mapped out in the preceding chapter and consider seriatim:—

- I. Pericarditis. Simple, malignant, and chronic sclerosing.
- II. Endocarditis. Simple, malignant, and chronic sclerosing.
- III. Myocarditis. Acute, simple, and malignant.
- IV. Rheumatic serositis.

I. ACUTE PERICARDITIS

(a) Simple

Pericarditis marks a severe infection. It occurs more often in childhood than in adult life, and, as a rule, is not an early manifestation, but supervenes in cases already damaged by previous rheumatic attacks.

The violence of the symptoms differs, and there are few more remarkable occurrences in medicine than the discovery of general pericarditis in a child who has suffered no pain, and who may have aroused no suspicion of heart disease, having come under notice for general weakness and wasting.

Pain over the precordium, *palpitation*, and *dyspnoea* are the most constant symptoms. There is a moderate *rise of temperature* to 100° or 102° F., and possibly higher. The aspect is *anxious*, and the face *pale*. In adults there may be much *mental distress*, but neither in adults nor in children have I met with the acute delirium described in most of the textbooks, except in those cases in which the pericarditis was complicated by chorea, or salicylate poisoning.

The attitudes adopted by the patient are various. Many children throughout the attack rest comfortably in any position; in adults orthopnoea is more frequent, and sometimes the patient leans forward on his elbows over a bed-rest, or turns over on his face. The *respiration rate* is in severe cases much increased.

A most troublesome *cough* with but little expectoration is met with occasionally, and is a source of great distress.

Pleurisy, again, is a very frequent complication, and there is as a consequence often considerable pleuritic pain.

Should the case run a down-hill course, *livid pallor*, *restlessness*, and *vomiting* are frequent symptoms, and the mind may wander in *asthenic delirium*.

Physical signs. The *pulse* is rapid, the wave ill-sustained, and the blood-pressure low. Later, in severe cases, it becomes irregular, and finally running in character.

The physical sign of paramount importance is *pericardial friction*.

This is a superficial scratching or rubbing sound which is

either both systolic and diastolic in time, or less frequently systolic only. It is usually first recognized at the base of the heart, but the area may rapidly increase until the rub is eventually heard over the entire precordial area. It is modified and diminished in intensity by gentle pressure with the stethoscope.

The impulse of the heart is displaced outwards, and is diffuse on account of the acute dilatation which invariably occurs, and for the same reason the area of the deep cardiac dulness is increased to the left, to the right, and upwards. In addition to the pericardial friction a *mitral murmur* is almost invariably present, for it is seldom that rheumatic pericarditis occurs without endocarditis or without sufficient cardiac dilatation to produce a relative incompetence of the valve. In some cases the sounds have a *cantering rhythm*, and this may precede the appearance of pericardial friction. The action of the heart is greatly excited.

Although for convenience of clinical description the division of pericarditis into the three stages (1) of onset and early friction, (2) of effusion, and (3) of resolution, was an admirable one, it must be discarded here because it gives a wrong impression. At the time when it was generally accepted, the large area of precordial dulness that is always found in severe pericarditis, was interpreted as evidence of an effusion into the pericardium; but since it has been demonstrated conclusively that this enlargement is almost entirely the *result of dilatation*, our views have been modified, and the retention of these stages now would give a false idea of the importance of effusion in rheumatic pericarditis. Although many cases have been under my observation, I have never seen one in which paracentesis pericardii was needed, and have never met with a fatal case in which this operation should have been undertaken, although I do not

deny that such a need may arise. Yet in many of these cases the area of cardiac dulness had been very greatly increased.

In a description of rheumatic pericarditis it is sufficient then to direct attention to the not infrequent occurrence of a slight *muffling of the cardiac sounds*, synchronous with the disappearance of pericardial friction, and to interpret this as the result of some moderate effusion into the sac, and to state that a large effusion may, though very rarely, occur. Often-times, however, no such change as this is noticed, but the area of cardiac dulness steadily increases with persistence of friction and with clearly audible cardiac sounds, in which case it is certain that any effusion there may be is negligible in quantity, and the progressive enlargement must be the *result of myocardial failure*.

When the dilatation is severe the liver becomes congested and enlarged from incompetence of the tricuspid valve, and some slight oedema of the ankles may appear. The lungs, too, suffer and become congested and oedematous.

In fatal cases death is sometimes sudden from *syncope*, or *more gradual* and preceded by great cardiac weakness, increasing dyspnoea, vomiting, restlessness, and a subnormal temperature. A subnormal temperature is repeatedly met with in severe pericarditis.

When there is *recovery*, the pulse and respirations become less frequent, the colour improves, and the dilatation diminishes. If the friction has previously disappeared it may reappear again for some days when the effusion is absorbed and the roughened surfaces of the pericardium come once more into contact.

The *duration* of an attack of pericarditis is very variable, but a case of average severity will be showing clear evidence of improvement in a fortnight.

Allusion has already been made to pleurisy. In some

cases of pericarditis the external surface of the pericardium and the opposing surfaces of the pleurae become inflamed and give rise to *pleuro-pericardial friction*. This sign differs from the pericardial in having a two fold rhythm, one cardiac, the other respiratory. This form of friction may occur without intra-pericardial friction, and is then of much less serious import than the latter: on this account it is essential to recognize its character.

(b) **Malignant Pericarditis**

Malignant pericarditis is characterized by the great severity of the symptoms, and it usually supervenes in cases that have already suffered from cardiac rheumatism.

The following is an example of the protracted type of this condition:—

A boy aged seven had suffered from cardiac rheumatism at the age of six, and three weeks before coming under observation had been ill with multiple arthritis and pre-cordial pain.

There was severe mitral disease, and there were also many rheumatic nodules.

His illness lasted $3\frac{1}{2}$ months, and throughout that time there was irregular fever varying between 104° and normal. No treatment was of any avail. He became progressively paler and more dyspnoeic, and at intervals pericardial friction appeared. The heart steadily enlarged, and was always excited. The liver enlarged, and eventually some oedema appeared. Finally, the child died from progressive heart failure.

The post-mortem examination showed old standing mitral and tricuspid endocarditis. The pericardium was greatly thickened, and the layers glued together by recent adhesions, with some exudation, at the base both in front and behind.

In the more acute cases the pericardium may be thickened in the same extraordinary way, and the *exudation be haemorrhagic*. Such cases run a truly malignant course in that the patient is profoundly toxic, ashen in colour, and never shows a reaction to any remedies. Just as in the non-malignant cases, towards the end of life the temperature may drop to subnormal.

(c) Chronic Sclerosing Pericarditis

This form is *not infrequent* in childhood. There are no violent symptoms, and there may be a *latency* which is most deceptive. The gravity of the condition lies in its persistence and its detrimental effect upon the muscular power of the heart.

The symptoms are: slight fever, precordial pain, and the occurrence of localized pericardial friction which, after lasting for some weeks, disappears, only to reappear later in another spot. In this way the illness may drift on for many months, the child eventually making a very imperfect recovery, or dying from gradual heart failure.

Such a condition may be complicated by *pleurisy*, and sometimes also by a *subacute peritonitis* around the liver and spleen.

When in such cases the heart becomes shackled by dense fibrous pericardial adhesion, there supervenes a remarkable train of symptoms which is described under the name of multiple rheumatic serositis.

THE DIAGNOSIS OF RHEUMATIC PERICARDITIS

This is not difficult on account of the frequency with which pericardial friction occurs, and that sign can be mistaken for few others. Occasionally a *double aortic murmur* is confusing, but this is not modified by pressure, the sounds too seem

further from the surface, and are usually blowing rather than scratching in quality.

Pleuro-pericardial friction differs in having both a cardiac and a respiratory rhythm.

Dilatation of the heart may prove a difficulty, but this is more often met with in other forms of pericarditis with much effusion. The distinctions are for this reason given on page 162.

The great practical difficulty in the diagnosis of rheumatic pericarditis is not so much in the differentiation of the condition as in the detection of its occurrence.

The pericardium may, for example, be adherent in front and free behind, and in this position there may be pericarditis. There are then cardiac excitement, dyspnoea, dilatation, and fever, *but no friction*. Such cases can only be guessed at from experience, for the physical signs that can be detected at the back of the chest are too equivocal to rely upon for the means of diagnosis.

On the other hand, difficulty may arise when the pericardium is partially adherent, the heart large and its action excited. Under these circumstances there may occur *indefinite, superficial, churning sounds*, which may lead to a diagnosis of acute pericarditis when there is no recent inflammation. The result of rest in bed usually settles this question, for in this condition, when the action of the heart quietsens, the churning sounds disappear, and the signs of acute pericarditis do not develop.

When pericarditis has been determined, the next step in the complete diagnosis is to decide upon the cause. There is seldom difficulty, for such other evidences as arthritis, nodule formation, erythematous rashes, or chorea are usually present.

Suppurative pericarditis may naturally be confused, but as

a rule there is no friction in these cases, and rather than being mistaken for the rheumatic form they are generally not diagnosed at all.

Tubercular heart disease may, however, closely simulate the rheumatic form, and when pericarditis occurs it is most difficult to differentiate them, for there may be definite friction in this form. Reliance must be placed upon the history of the case, and upon manifestations of active tuberculosis in other parts of the body.

II. ENDOCARDITIS

(a) Acute and Subacute Simple Endocarditis

Just as rheumatic fever is the most frequent cause of pericarditis, so it is also of simple endocarditis.

The symptoms are unobtrusive, and were it not for the frequent occurrence of other manifestations of rheumatic fever endocarditis would repeatedly escape notice. We have only to realize the minuteness of the lesions to understand this difficulty, which has led to the establishment of a cardinal rule of practice.

In all cases of rheumatism examine the heart.

The symptoms that usually attract attention are :—

1. Vague pains over the precordium.
2. Palpitation.
3. Shortness of breath on slight exertion.
4. Slight fever.
5. Pallor ; and later, wasting.

The *physical signs* depend upon the valve affected, although in all cases there will be some dilatation of the heart from the rheumatic damage to the myocardium.

(a) Endocarditis of the mitral valve. This produces

mitral regurgitation, the *first sound* in the region of the impulse alters in character, and is *roughened* or shorter in duration than usual. More significant still is the appearance of a *systolic murmur*, soft and blowing in character, which can be traced toward the left axilla.

This bruit may not at first be audible with every heart-beat, and in childhood it is frequently noticed later that this systolic murmur alters to a *to-and-fro bruit*.

In a comparatively short space of time the mitral murmur may become loud and musical. Another physical sign of importance is the appearance of a double sound at the impulse, which resembles in character a reduplicated second sound, the mental effect it produces is verbally represented by *lub-tüt-tüt*. This sign, if it is persistent, probably indicates a definite thickening and imperfect mobility of the cusps of the mitral valve.

There will be the usual evidence of acute dilatation, and later, if the incompetence is of any moment, signs of hypertrophy will appear, and the condition drifts into one of chronic valvular disease: vide Chapter viii.

The pulse at this early stage is increased in rate, and the wave compressible, but the mitral characteristics will not yet have developed.

(b) **Endocarditis of the aortic valve.** This produces aortic regurgitation.

The onset is even more insidious than in endocarditis of the mitral valve. The general symptoms are the same, but the special signs are: a suspicious *feebleness* of the *aortic second sound*, then sometimes the appearance of a *systolic murmur* over the aortic region immediately following the first sound, and finally the appearance of the characteristic *diastolic murmur*. This bruit is at first short, soft, and faint, and this lesion is often overlooked.

In contradistinction to mitral regurgitation the characteristics of the aortic pulse soon make their appearance.

These characters are :—

(1) Increase in rate.

(2) Alteration in the character of the wave, which becomes large, sudden, and collapsing.

(3) The development of capillary pulsation.

There is the usual acute dilatation of the heart associated with active rheumatism, followed later by the development of hypertrophy to compensate the cardiac embarrassment caused by the damaged aortic valve.

The *diastolic murmur* of early aortic regurgitation may be overlooked, not only on account of the softness, but because the site at which it may be detected varies. Sometimes this is the second intercostal space on the right side, close to the margin of the sternum. In childhood it may be the third left intercostal space, close to the sternal margin. Again, it may only be detected behind the body of the sternum.

(c) **Endocarditis of the tricuspid valve** results in tricuspid regurgitation.

Rheumatic inflammation of the tricuspid valve is seldom sufficiently severe to lead to detection, and in the presence of an invariable and much more evident mitral lesion is the more liable to be overlooked.

The physical sign upon which most reliance can be placed is the appearance of a systolic murmur, soft and blowing in character, audible over the lower end of the sternum, and in the fourth and fifth intercostal spaces on the left side close to its edge, and disappearing when the stethoscope is moved toward the impulse.

(d) **The pulmonary valve.** Acquired endocarditis of this valve is so rare that it will not be considered here.

(e) **Combined acute and subacute lesions of the valves.** The most important of these is a combination of mitral and aortic endocarditis. Its frequency is in part due, I think, to a *direct infection* of the aortic valve from the mitral.

In most cases the *aortic lesion follows the mitral* either in the same illness, or during another attack. Whenever a case of rheumatic endocarditis of the mitral valve remains obstinate, with the temperature continually raised and the action of the heart excited, and yet there is no pericarditis, a very close watch should be kept upon the aortic region, for it is in cases such as these that the disease spreads and implicates the second valve. The condition in this active stage is very closely allied to malignant endocarditis.

Such symptoms as pallor, fever, dyspnoea, cardiac dilatation, and palpitation, are usually more severe than when a single valve is attacked, and are likely to be more definite.

Two distinct bruits will be detected, the mitral systolic at the impulse, and the aortic diastolic at the base.

The character of the pulse will greatly depend upon which of the lesions is predominant. If it is the aortic, then the pulse will be collapsing, but if it is the mitral there may be no distinctive character.

These multiple valvular lesions occur more frequently in the young than in the adult.

The course of simple endocarditis, so far as recovery from the active lesion is concerned, is favourable, and slight mitral and aortic lesions may, after a while, completely disappear. Unfortunately the usual result will be a certain amount of *permanent damage*, a consideration of the effects of which, in Chapter viii, will form the basis of the study of chronic valvular disease.

It is impossible to state exactly from the physical signs

alone when a simple endocarditis commences, and equally impossible to be sure when the lesion is healed.

We are compelled to take into consideration the general condition of the patient, and judge from a review of the *entire case*. It is the difficulty in ascertaining the exact condition of a damaged valve which forms one of the greatest obstacles to the successful treatment of endocarditis.

**(b) Rheumatic Malignant, Pernicious, or
Ulcerative Endocarditis**

Some authorities do not accept a malignant form of endocarditis as rheumatic, but believe that such an occurrence in rheumatism points to a *secondary infection* of the valve or valves with 'septic' micrococci. The evidence in support of the position adopted here has been given upon page 86, and from this it will be readily understood that there must be often great difficulty in detecting the development of the malignant type of endocarditis in rheumatism.

It is more frequent in *early adult life* than in either childhood or maturity, and *predisposing* to its occurrence are: previous attacks of endocarditis, anaemia, mal-sanitation, especially if in combination with damp; a return to an arduous occupation when cardiac rheumatism is still active, and the anaemia produced by the disease uncorrected. Pregnancy is also probably a predisposing factor.

The chief symptoms that are of value in the diagnosis are:—

I. General evidences of severe constitutional poisoning, such as:—

- a. Irregular fever.
- b. Rigors. These are unusual in the rheumatic form.
- c. Sweating and rapid wasting.
- d. Progressive anaemia.

e. Diarrhoea.

f. Purpura.

g. Delirium.

II. Local manifestations:—

1. A persistently excited action of the heart.

2. Variability in the character of the cardiac bruits.

3. Evidence of infarction.

1. It is a rule, with few exceptions, that with active endocarditis the heart is increased in rate and its action excited.

2. The variability of the cardiac murmurs is not a constant feature, but when it is present it is a valuable sign. Its occurrence is dependent, sometimes, upon the presence of exuberant vegetations, which may suddenly be removed by the tearing of the vegetation, and sometimes upon the ulcerative process which may suddenly cause a rupture or perforation of a valve-segment.

3. Infarction has been considered in Chapter iv.

(c) Chronic Sclerosing Endocarditis

The chronic sclerosing forms of rheumatic endocarditis may produce mitral, aortic, or tricuspid stenosis, but when there is tricuspid stenosis this is combined with mitral, and sometimes with both mitral and aortic stenosis.

This type of inflammation forms a link between the acute conditions already considered, and chronic valvular disease of the heart into which it insensibly drifts.

MITRAL STENOSIS

The commencement of the affection often dates from childhood, and it is frequently associated with stubborn and obstinate *chorea*.

There may be no history of rheumatism, and the condition

may be discovered by chance to the surprise of the patient who has been unconscious of any heart affection. Such an occurrence is not difficult to understand when we remember how, in childhood, an attack of cardiac rheumatism may be accompanied by only a few vague pains and some malaise.

The earliest *symptom* is, as a rule, shortness of breath on exertion.

The *early physical signs* need careful study, for they differ in different cases.

In most cases, as would be expected, there is some evidence of *mitral regurgitation*, for it is difficult to imagine the segments thickened, and the mitral ring contracted, without some incompetence due to the impaired mobility of the valve, and as a result of this, there is usually, but not invariably, a systolic mitral bruit. A very suspicious sign is the constant appearance of an *apparently reduplicated second sound*, represented as *Lub-tut-tut*, which is strictly limited to the region of the impulse. Gradually this sound becomes more rumbling and longer in duration, until it develops to an obvious *presystolic murmur* accompanied by a *presystolic thrill*. The systolic murmur may then become fainter and disappear, and there are left a presystolic murmur, first and second sound. The second sound in the pulmonary region is accentuated.

In other cases the first murmur to be detected is the *presystolic*, and no systolic murmur is heard at any time.

In yet other cases the rumbling murmur is *diastolic* in time rather than presystolic, or it may be *mid-diastolic*.

Lastly, mitral stenosis may occur *without a definite murmur*, but with a remarkably sharp, short first sound at the impulse.

As the disease progresses the *aspect* of the patient becomes characteristic. The cheeks show a persistent red

flush, the result of the distended blood capillaries, and the lips are full and purplish. *Dyspnoea* increases, and cough is liable to be troublesome from *bronchitis* or constant high pressure in the pulmonary capillaries. These vessels for the same reason may rupture and *haemoptysis* result. *Palpitation* of the heart may now commence to give trouble, and these patients are often troubled with *subacute rheumatic attacks*. Sir William Broadbent has pointed out certain changes in the physical signs which are in some measure a guide as to the extent of the stenosis, and to the gravity of the condition. They can be but a partial guide, because one patient may have a powerful heart and constitution and a considerable lesion, and another, who is in a more serious condition, may have a slight lesion, but a feeble heart and constitution.

The physical signs are grouped by him into three stages; in the first stage there are :—

(1) A presystolic thrill.

(2) A presystolic murmur, and first and second sound at the impulse.

(3) An accentuated pulmonary second sound at the base.

In the second stage :—

(1) The presystolic thrill is more evident.

(2) The presystolic murmur is longer and louder.

(3) The second sound disappears in the region of the impulse.

(4) The pulmonary second sound is still more accentuated.

The explanation that is given to account for the disappearance of the second sound at the impulse is as follows :—

The second sound normally heard there is the aortic second sound only, but when there is much stenosis the right ventricle hypertrophies and displaces the left ventricle from the impulse, and thus abolishes the second sound.

It is, however, difficult to understand why under these

circumstances the pulmonary second sound does not become audible.

The pulse in this stage is often very characteristic, being strikingly small in volume, and the wave not easily compressible. The change from the first to the second stage indicates that the lesion is still smouldering on, for it points to increased stenosis and to increased effort at compensation.

The third stage is the *stage of failure*. The right ventricle gives way, and the physical signs once more change.

(1) The presystolic thrill disappears.

(2) The presystolic murmur disappears.

(3) This leaves at the impulse only a short, sharp first sound, with no second sound.

(4) The pulmonary pressure falls, and the second sound in the pulmonary region is no longer accentuated.

The condition is now one of chronic valvular disease, for as a rule the active disease has died out, leaving behind this irreparable damage. This final stage is considered under chronic valvular disease, p. 183.

AORTIC STENOSIS

This is much less frequent, and in childhood a very exceptional lesion.

There is the same history of protracted rheumatism, and the same liability to slight relapses as in mitral stenosis, but the physical signs differ in the appearance of a *systolic basal murmur*, with its point of maximum intensity over the aortic cartilage. This murmur is at first soft, but usually becomes harsher as the lesion progresses, and it is often accompanied by a *thrill*. The left ventricle slowly *hypertrophies*. A fuller consideration of this lesion, and of the still more rare *tricuspid stenosis*, is given under the chronic valvular lesions, pp. 139 and 142.

III. MYOCARDITIS

(a) Acute

In the preceding chapter it was pointed out that myocarditis was not an altogether satisfactory term, for in many cases there is in all probability no actual myocarditis, but rather a damage to the contractile power of the cardiac muscle by the rheumatic poisons.

Be this as it may, the clinical signs of acute dilatation of the heart are among the most important in the entire range of heart disease, for *they afford the earliest evidence we possess of rheumatic heart disease.* This part of the subject demands then the closest study.

There are three clinical types:—

1. Acute simple dilatation.
2. Acute malignant dilatation.
3. Chronic dilatation.

Such distinctions are in some degree arbitrary, but they have also a certain practical value.

1. *Acute simple dilatation.* For our knowledge of this we are greatly indebted to the writings of D. B. Lees, although in 1870 Gull had already taught the importance of this rheumatic dilatation.

Symptoms. The symptoms are few, and the condition is easily overlooked.

Some *breathlessness, pallor*, and a *rise of temperature* co-incident with the rheumatic attack, are the usual indications.

Physical Signs. Its detection depends upon a careful physical examination of the heart.

There is an increase in the frequency of the *pulse*, and the blood-pressure is low.

The *impulse of the heart* moves outward, and is feeble. The deep *cardiac dulness* is increased both to the right and

the left. Upon auscultation it is noticed that the *first sound* is short and the *second sound* in the pulmonary region is accentuated, and sometimes reduplicated. Finally, in some cases a *soft systolic murmur* appears, with its maximum intensity internal to the left nipple line.

This dilatation may sometimes supervene with great rapidity while the patient is under observation.

In a considerable number of cases there may be *complete recovery*, and in some three weeks from the time of onset it will be found that the systolic murmur has disappeared and the heart has regained its usual dimensions. In this class must probably be grouped a certain number of the examples of 'cured' mitral endocarditis.

Unfortunately *dilatation* and *mitral endocarditis* often co-exist, and in such cases, although there will be recovery from the acute rheumatic dilatation, there will also probably be some necessary dilatation and hypertrophy in order to compensate the valvular lesion.

2. **Acute malignant dilatation.** This is one of the rarest occurrences in rheumatic heart disease. It is a virulent process and rapidly produces a fatal failure of the heart from profound myocardial poisoning. The symptoms closely resemble those of a severe pericarditis, but there is no friction. The patient is *breathless* and *livid*—there may be *syncopal attacks* and *delirium*. The *pulse* is rapid, feeble, and irregular. The impulse of the *heart* is diffuse or lost altogether, the action excited, the area enlarged, and the sounds short and clear. A systolic mitral murmur may be present. In one such case of a more subacute type recorded in my clinical lectures on rheumatic fever, I found after death two projections on the surface of the right ventricle resembling the blister-like excrescences upon the bulb of an overstrained Higginson's syringe. At these spots there was practically

no sound muscle at all, the aneurysmal visceral pericardium alone intervening between the blood in the right ventricle and the pericardial cavity.

IV. MULTIPLE RHEUMATIC SEROSITIS

It is difficult to group this particular condition, but it may be conveniently described here.

The condition is a rare one, though of importance because of its great gravity. It is essentially a chronic smouldering, relapsing inflammation of *several* serous membranes.

Morbid anatomy. I have on occasions made necropsies upon severe cases of rheumatism in childhood, in which, in addition to acute pleurisy and pericarditis, there has also been *local peritonitis* around the liver and spleen, and it is remarkable that the clinical condition of multiple serositis is not more frequent. When it does occur there is a group of symptoms, the exact development of which depends upon the particular serous membrane which first shows the stress of the affection. This in most cases is the *peritoneum* only, for the heart is embarrassed by the adhesions and produces back-working, the inflamed peritoneum weeps, and the peritoneal cavity is so extensive that it cannot readily be obliterated by adhesion, as may happen with the pericardial and pleural cavities. In addition there must be taken into consideration the possibility of cirrhotic changes in the liver, and partial obliteration of the right auricle or inferior vena cava by pericardial adhesions.

The symptoms may develop some months after a very definite attack of rheumatic fever. In other cases the onset is most insidious, the original rheumatic attack being quite subacute.

Symptoms. *Ascites disproportionate to oedema, swelling of the face, dyspnoea* and *cyanosis* are the usual symptoms.

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Physical signs. The physical signs are in well-marked instances very characteristic. The *cardiac impulse* is diffuse, feeble, or lost, the *area of dulness* is enlarged, and the cardiac sounds feeble and muffled. The *veins in the neck* are full, and the *pulse* rapid and feeble. Examination of the chest may discover *signs of fluid* at one or other base.

When the fluid is removed from the abdomen a *large smooth liver* is felt.

The interpretation of the signs will be an adherent pericardium, chronic pleurisy, and probably also some chronic peritonitis.

The course of the illness is prolonged, and although the mild cases may quiet down, more usually there is a history of repeated tappings, now of the abdomen, now of the pleurae. Fresh outbursts of inflammation occur, and pleurisy or a patch of pericarditis may be detected; the wasting becomes extreme, and eventually all the signs of complete cardiac failure may develop.

The diagnosis in such cases is easy. When the symptoms and signs are ill-marked the condition may be most puzzling, and may never be diagnosed during life. The further consideration of this question is deferred to the section on tuberculosis of the heart, p. 176.

The treatment is palliative, prolonged rest and open-air treatment affording the best chance. A rest in a hospital sufficient to establish compensation is of little use, for renewed physical exertion at once disturbs this balance. Many months of rest after the disappearance of the ascites are necessary. Obliteration of the peritoneal cavity is seldom indicated on account of the condition of the heart.

CHAPTER VII

PROGNOSIS AND TREATMENT OF ACTIVE RHEUMATIC HEART DISEASE

Prognosis of pericarditis—Endocarditis—Myocarditis—Treatment—
Prophylactic—Immediate—Pericarditis—Palliative method—Specific
method—Treatment of Endocarditis—Simple—Malignant.

1. The Prognosis of rheumatic pericarditis. In making the prognosis there are two distinct aspects to be considered; the first, the *immediate outlook* as to life or death; the second, the condition of health that is to be expected in the *future*, when recovery from the attack is complete.

(i) The immediate prognosis is as a rule good with these exceptions:—When the type is malignant. When the pericarditis attacks a heart already badly damaged by rheumatism, or when it supervenes after a severe chorea. Lastly, the chronic and relapsing type is of a serious prognosis, for a considerable number of them prove eventually fatal without any real intermission of the symptoms.

(ii) The prognosis as to the future is beset with difficulties. When a pericarditis, however mild, occurs in a child under ten years there is always the unknown factor of the possibility of another attack of cardiac rheumatism.

Upon this point, in the present state of our knowledge of rheumatism, we have no guide to direct us.

A mild pericarditis does well if only sufficient time is given for the convalescence, and there are sometimes wonderful recoveries that leave no apparent traces of the attack.

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When, in spite of prolonged care and rest, the heart remains rapid in action and dilated, and there is evidence of pericardial adhesion, the outlook is bad. Equally serious are those insidious sclerosing forms of pericarditis in which after an interval dropsy commences with cyanosis, dyspnoea, and enlargement of the liver.

2. **Endocarditis.** *The immediate prognosis of simple endocarditis is good ; it is, in fact, never directly fatal. The remote prognosis is considered under the general prognosis of chronic valvular disease.*

The *malignant form* is very fatal, and when all the chief symptoms are present recovery is quite exceptional. There are, however, transitional cases in which the fever subsides and after a long illness there is imperfect recovery. In these the symptoms are often equivocal: infarctions are few, the general state of the patient stationary rather than progressive, and the temperature, though persistent, not of wide range.

3. **Myocarditis.** *The prognosis in virulent myocardial disease is almost hopeless, but the subacute cases recover, though often slowly, and it can hardly be claimed that this point has yet been worked out. Simple acute dilatation often recovers completely.*

4. **Multiple serositis.** This is of uniformly gloomy outlook.

5. **Relapsing carditis** is also a most serious condition, ending frequently in a general rheumatic toxæmia.

THE TREATMENT OF ACTIVE RHEUMATIC CARDITIS

(a) The Prophylactic Treatment

Rheumatic fever is so much more frequent in the poorer classes than it is in the well-to-do, that one feels that there must certainly be much that is preventable in its causation.

The demonstration of its infective origin points still further in the direction of prophylaxis. Although in some measure this disease must be preventable, it is not, in my opinion, by any alteration of diet. I have signally failed to discover any particular error in the everyday diet of rheumatic children.

The influences of crowded towns and board schools, of damp and jerry-built houses, of malsanitation, soil, and climate, are problems which would well repay re-examination with the infective origin of the disease in view.

The heredity of the disease and its incidence in several members of a family should warn us to explain thoroughly to parents, guardians, and patients the true meaning of repeated sore throats, vague pains, chorea, erythematous rashes, and general failure in health among the predisposed. For some years I have kept a number of rheumatic children under regular supervision, and though there have been many disappointments I believe that some little good has been done by precautionary measures. Nevertheless, with rheumatism as with tubercle there is this great difficulty, that one never knows when the disease is only latent and not really cured.

(b) Treatment of Active Rheumatic Morbus Cordis

In the treatment of active rheumatic disease *rest* is the first and most important step. These patients should be protected from draughts.

The *food* should be liquid, and consist chiefly of milk suitably diluted, or made more digestible by adding *citrate of soda* in the proportion of a grain to the ounce. When the attack is severe it may be necessary to feed the patient most carefully every two hours in the day and every three

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hours at night with peptonized milk or even asses' milk. Unless there is clear indication that the kidneys are also damaged, there is no objection to well-made beef tea, chicken or veal tea.

When the attack though active is not severe, and the digestion is good and the temperature little if at all raised, the diet should not be so restricted, and in the lingering relapsing cases in childhood better results are obtained *from a liberal diet* in which fish, sweetbread, chicken, and underdone mutton are allowed ; custards, jellies, milk puddings are also indicated. I think there is no serious support for the belief that *nitrogenous foods* have any harmful effect in rheumatic heart disease, provided that the general condition of the patient is suitable for their employment. It is on the other hand a decided gain where in heart affections, with their tendency to gastric atony, small quantities of solid food can be given. *Stimulants* are not needed as a routine, but they are valuable aids when the pulse is feeble, the appetite flagging, and there are general feebleness, pallor, and restlessness. Brandy is the most useful, but whisky and champagne are also of value not only to make variety, but as alternatives when brandy is disliked.

A child of seven years can begin with half an ounce of brandy in the twenty-four hours, an adult with two ounces.

Much has been written about the detrimental effects of alcohol upon the cardiac muscle, and its power to produce vaso-dilatation, but used for a short time of need it seems impossible that it can do harm, and of its value as an aid to sleep and digestion there is surely no doubt.

The *clothing* of these patients should be warm and soft, for they are often unable to lie down in bed, and so are liable to get chilled. The garments should also be loose and easy, and particular attention be given to the protection of the lower

part of the neck and arms. Hot-water bottles and water beds ; all the accessories, in fact, of skilled nursing may be needed in difficult cases of acute rheumatic heart disease.

Medicinal treatment. This must at once raise the debated problem of the use of salicyl compounds. There are two questions to be answered :—

- (i) Are these drugs specific in rheumatism ?
- (ii) Can they be safely pushed ?

The second question must be answered, because there is general agreement that small doses cannot cope with cardiac rheumatism.

The answer to the *first question*, given by many authorities is 'Yes', but I am in accord with those who answer in the negative.

The *second question* is an easier one, for any drug that has a powerful action must have also a limit dose, and this limit, differing doubtless for individuals, must eventually be decided upon by those who employ the drug largely.

In recent years Lees has certainly demonstrated that, with improvement in the drugs, the limit of tolerance is higher than was generally believed.

There are then at the present time two different lines of treatment of acute rheumatic heart disease, and both of these will be given here. The first is the *palliative* treatment, the second is the *specific treatment*.

1. The Palliative Treatment

This method keeps in mind that there is a great *natural resistance* to the rheumatic infection, and it attempts to aid this in every possible way.

The chief principles of this method are illustrated by the treatment of a severe attack of acute pericarditis. As a

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routine no application *eases the pain* more effectually than an *ice-bag* applied to the precordial region. The steady advocacy of this method by Lees has gained many converts. The pain is generally relieved, and the heart quieted. The constant application is most serviceable, but this needs at least one, and preferably two skilled nurses, and for the first twelve hours the temperature should be taken every two hours, after this at longer intervals. Strict orders should be given as to its use, and the area indicated by the medical attendant. Two bags are advisable, so that when one is used the other is ready for application. They are liable to leak unless the caps are carefully screwed down, and the ice must be carefully pounded to prevent injury to the rubber. Nothing but the finest gauze should intervene between the bag and the chest wall, and if the full effect of the ice is feared on account of the weak condition of the patient it is better to abandon its use and trust to other means. When there is much precordial tenderness the bag should be slung so as to bear lightly on the chest. With children the best plan for fixing the ice-bag is to make a hole in the vest, through which the narrow part of the bag is passed, and then fastened by a flannel band passing round the neck. The lower border of the vest is fastened to a binder.

Should there be a rapid fall of temperature and signs of collapse the ice-bag must be promptly removed, and in all cases hot-water bottles are placed near the lower extremities.

The use of ice is contra-indicated in asthenic cases with a normal or subnormal temperature.

In some cases of pericarditis the acute dilatation may produce a condition of urgent danger from embarrassment of the right side of the heart. The face is livid, the cardiac dulness to the right of the sternum much increased, the

right ventricle impulse forcible and struggling, and the radial pulse small and easily compressible. Dyspnoea and restlessness greatly distress the patient. In such cases *blood-letting* is of value. Rheumatism is so destructive to the blood that there is natural hesitation to adopt this procedure, but experience shows that prompt leeching may do good, and a strong adult can be bled without fear of doing harm under these particular circumstances.

Four leeches can be applied to the pericardium in childhood ; for an adult six or eight leeches can be applied, or *ten ounces* of blood may be taken if venesection is decided upon. These are exceptional occurrences, and in most cases of this dilatation *cardiac stimulants* will answer the purpose.

If the ice-bag cannot be tolerated, and yet there is much pain and precordial tenderness, a *blister* applied over the most painful area often gives relief, but this again is not advisable as a routine measure. If there is only discomfort, *hot wool* or *light poultices* are comforting, and the proprietary application *antiphlogistine* is cleanly and helpful.

Internal remedies. The bowels should be opened freely at the onset, but purging later should be avoided as unnecessary and weakening. *Calomel*—one grain for a child and three for an adult—should be followed by a morning dose of *magnesia*, or a *Seidlitz powder*.

If there is pain and suffering from concomitant arthritis *salicylate of soda* (1) combined with *bicarbonate of soda* and *nux vomica* should be given, with the intention of relieving the pain.

Many cases of pericarditis need no special measures, and then *quinine* (2) given in an effervescent form to adults, in simple solution (2) to children, is a safe routine.

For *restlessness and insomnia with distress* there are various remedies recommended, and of these I prefer a mixture of

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nepenthe and bromide (3). *Nepenthe* disturbs the digestion less than most of the forms in which opium is prescribed, and both adults and children are often much relieved by this prescription. *Trional* in fifteen-grain doses, *veronal* combined with bromide in five to seven-grain doses, and *paraldehyde* (4) in drachm doses, are all used for insomnia and *restlessness without pain*, and in some cases they answer well.

When the *left side of the heart is failing*, and the action rapid and feeble, *digitalis* is indicated unless there is reason to believe there is a large effusion. It must be admitted that its effects in these acute inflammatory lesions are not particularly encouraging; by small doses (5 to 7 minims of the tincture), however, the heart is often steadied. The tincture may be used or the granules of digitalin, which are a convenient and accurate method of presenting the drug. *Strophanthus* in the same dosage is preferred by some (5). *Strychnine* is also valuable when feebleness rather than rapidity indicates cardiac failure, but it is a mistake to have recourse to this drug early in the illness when the heart is excited, for the action is then still more excited by the drug and the heart thus worn out. It is best given *by the mouth*, and should only be used hypodermically when there are pressing indications of cardiac weakness. Neither strychnine nor digitalis should be left off abruptly, for the heart misses them, and it is a mark of clumsy treatment to press them to such a point of intolerance that they must be abandoned abruptly.

An *ammonia and ether mixture* (6) is a valuable restorative for *sudden collapse*.

All *gastric symptoms* must be attended to promptly, the milk peptonized, or stopped for twelve hours and teaspoonful doses of concentrated meat essences substituted.

Sometimes nutrient enemata may be needed. If there is much gastric irritability full doses of *bismuth and pepsine*

after meals are indicated. A *pleural effusion* must be dealt with early in all cases of heart disease.

Lastly, it is better to do too little than too much for rheumatic pericarditis. The best recoveries I have seen have been without the use of salicylates, but with good nursing and careful dieting; and the repeated disturbance of children for medicine, food, and temperature taking is detrimental to their recovery.

Acute endocarditis is generally present with the pericarditis, or it may occur irrespective of it. In either case there is no special treatment; although some definite lines of procedure should always be confidently adopted. Rest, small doses of *quinine* (2), and an ice-bag if the heart is excited, are indicated. *Prolonged rest* is generally admitted as the most important factor in the treatment of acute valvular disease, but all medical men know the difficulties with patients, and wrong impressions that may arise, in a condition where there are so few severe symptoms. It is for this reason that a confident and definite line of procedure should be adopted from the first, and in this way the help of the patient or the parents obtained for at any rate part of the treatment.

2. The Convalescent Stage of Carditis

Important guides as to its progress are obtained from a continued normal temperature, absence of rheumatic signs, improvement in the pulse and heart sounds with diminution in the cardiac area. These are preferable to any time limit, for the individual cases vary greatly. Cardiac tonics should be gradually withdrawn and replaced by *quinine and arsenic* (7) in alkaline solution. *Iron* should be given with caution. The resting-stage can be well employed in the using of *passive movements*, by which the heart is strengthened and prepared

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for voluntary exercise. Each forward step should be gauged by temperature, pulse, and heart.

As soon as possible, removal to a warm, sunny, dry climate is advisable, where *open-air treatment* and *liberal diet* will replace drugs. This country, where rheumatism is so severe, should have homes in connexion with the great hospitals in which the numerous cases of acute rheumatic carditis could prolong their convalescence.

Exercise should begin cautiously, along the flat at first and then up gentle inclines. After severe carditis, by these measures great improvement may be effected in six months' time, and although there is frequently an organic valvular lesion remaining, the prolonged rest will have enabled it to heal. It may be pointed out that the rest advised is a purposeful one, and the physician is always trying to get the patient forward, carefully testing the power of the heart, and training it for a return to ordinary life.

Such careful measures can rarely be adopted, but nevertheless an effort in this direction can be made even if the means or scepticism of the patient make its accomplishment quite hopeless. This at least is certain, that at the present time there is no rapid cure for severe rheumatic carditis, and without doubt the lesions take a long time to heal.

3. The Specific Treatment of Rheumatic Carditis

This has been recently detailed by Lees, who firmly maintains with other authorities that the salicyl compounds are a *specific* in rheumatism.

For an adult twenty grains of sodium salicylate are combined with forty of sodium bicarbonate every two hours throughout the day, and every four in the night. Children from six years onwards may commence with half this dose, but in proportion they require larger ones than

adults, and accordingly after a day or two the dose is raised. Should vomiting or any untoward symptom arise the drug may be suspended for a few hours and then commenced again in smaller doses, and combined with some *aromatic spirits of ammonia*.

The maximum dose for both adults and children is, in severe cases, thirty grains of salicylate of soda, sixty of bicarbonate of soda.

The drug should be persisted in for some while after the subsidence of the active inflammation.

It is clearly important to recognize the symptoms of salicylate poisoning if this method is used.

These are :—

1. Delirium, tinnitus, deafness, vertigo, and headache.
2. Intense general depression.
3. Vomiting, which is incessant and sometimes difficult to arrest.
4. Polyuria.
5. Slow, feeble pulse.
6. Air hunger. Collapse and death.

Those who undertake this method of treatment should be fully aware of its dangers, slight though they may be in skilled hands, and with drugs of the very best quality, for they may under less favourable circumstances introduce new dangers. The natural salt is still the best preparation of the salicyl compounds, but its expense is a great bar to its use in large doses over a considerable period of time.

MALIGNANT ENDOCARDITIS

Treatment is very unsatisfactory, but I firmly believe that in the rheumatic this disease can be to some extent prevented if these points are remembered.

- (1) That a patient allowed to go back to work in stuffy

rooms with a partially healed simple rheumatic endocarditis runs a risk of the graver disease.

(2) That anaemia in the adolescent rheumatic should be carefully treated with mild iron preparations and arsenic, fresh air, sunshine, and rest.

(3) That rheumatic patients should be plainly told the dangers of sore throat, damp and insanitary surroundings, and the importance of the early warnings of fresh attacks of rheumatism.

(4) That it is very probable that the rheumatic infection may linger in a more or less latent form in the cardiac vegetations. This will put the medical man upon his guard when any general failure of health occurs in patients who are the subjects of rheumatic heart disease even before the definite manifestations of active rheumatism make their appearance.

General treatment. Unless these patients are too ill, *open-air treatment* should be undertaken, particularly if the season of the year is favourable.

I cannot convince myself that *Salicylates* are serviceable, but it is obvious that those who hold to their specific value would, if they accept the malignant rheumatic form of endocarditis, press them persistently in such cases.

Attempts with an anti-rheumatic serum are not yet encouraging, but these are in their infancy. I should in these cases try a *polyvalent anti-streptococcus serum* rather than abandon the case as hopeless, because it appears to me that we have no accurate knowledge of the various streptococcal poisons, and in these closely allied infections the same poisons may to some extent co-exist. Further it is not yet possible to determine accurately when we are dealing with mixed infections. The other remedies are given under the non-rheumatic forms of malignant endocarditis.

CHAPTER VIII

CHRONIC RHEUMATIC HEART DISEASE

Adherent pericardium—Pathology—Symptoms—Diagnosis—Prognosis—Treatment—Chronic valvular disease—Mitral regurgitation—Mitral stenosis—Aortic regurgitation—Aortic stenosis—Tricuspid regurgitation—Tricuspid stenosis—Pulmonary regurgitation and stenosis—Chronic rheumatic dilatation.

A. CHRONIC PERICARDIAL DISEASE

By this is meant the condition of adherent pericardium which is left after attacks of rheumatic pericarditis. This differs from multiple serositis in that the active process is dead, and there are only the mechanical results of adhesion left behind, which may be compared to the far more frequently occurring chronic valvular lesions.

I cannot do better than classify the chief forms of pericardial adhesions after the method of F. Roberts:—

(1) Partial and small adhesions between contiguous surfaces of the pericardium.

(2) Extensive internal adhesions (a) with thickening ; (b) without thickening.

(3) External adhesions only (rare) : this is a mediastinal sclerosis.

(4) Internal and external adhesions combined (the most important group).

(5) Marked pericardial thickening without adhesion (very rare).

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Results of pericardial adhesions. There seems agreement that general adhesion of the two layers of the pericardium without thickening or external adhesion produces little or no detrimental effect upon the heart; when on the other hand there are great thickening and external adhesion the heart suffers greatly.

The usual results are *dilatation and hypertrophy* of the heart. John Broadbent maintains that the heart is dilated during the acute pericarditis and caught, as it were, in this condition by the organizing pericardial adhesions. Unable for this reason to completely recover its normal size, and impeded by the adhesions, it then hypertrophies to cope with the maintenance of the circulation.

In arriving at any conclusion upon these points those cases must be chosen, exceptional ones though they are, in which there is no valvular lesion to complicate the problem. Under such circumstances there appears no doubt that hypertrophy and dilatation may result, and whether Broadbent's view explains all the cases or not, it seems reasonable to suppose that the shackling of the heart's free movement would lead to hypertrophy. On the other hand, importance must be attached to the particular process of inflammation which has produced the adhesion. If it is very chronic and massive the heart may be *strangled* rather than shackled, and then the muscle atrophies. This is a less frequent but a most serious event.

Lastly, the thin-walled *auricles*, particularly the right one, may be *compressed* by adhesions, when the ventricles have either escaped or are hypertrophied. In such cases the openings of the large veins may be seriously contracted.

In summary it may be stated that an adherent pericardium

- (1) May be latent;

- (2) May produce dilatation and hypertrophy of the heart ;
- (3) May produce atrophy of the heart ;
- (4) May produce local compression of the auricles, one or both.

Symptomatology. It will be evident from the preceding account that the symptoms must vary considerably in different cases, both in their character and mode of development.

Among the general symptoms are *pain, palpitation, and dyspnoea.*

When the right auricle is compressed there may be a *slowly progressive oedema* which may reach a most extreme degree, and implicate both the peritoneal and pleural cavities.

Physical signs. *The pulse* may show the characters of the *pulsus paradoxus*. In connexion with the *heart* the following signs have been recorded. 1. *Fixation of the apex* in an abnormal position. This sign is of no value in children when the heart is large, for there is no room in their chest for it to move with alteration of position, although there may not be a single pericardial adhesion.

2. *Systolic retraction* of the lower end of the sternum and adjacent costal cartilages.

3. *Systolic retraction* of the postero-lateral walls of the lower part of the thorax as described by John Broadbent is very suggestive, but not pathognomonic, for he points out that it may occur when the pericardium is not abnormally adherent to the heart, but is to the central tendon of the diaphragm.

4. *Systolic recession* of the chest in the region of the apex-beat.

5. *Enlargement of the precordial dulness* with unusual resistance to percussion and muffling of the heart sounds may

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be met with in cases where the pericardium is greatly thickened.

6. In children J. Broadbent lays stress upon the occurrence of a *presystolic murmur* of somewhat indefinite character which is not associated with mitral stenosis.

7. *Hampered respiratory movements*, of which the most important is impaired mobility of the diaphragm.

The diagnosis is to a great extent a matter of conjecture. I have paid particular attention to this point in childhood, and am convinced that it is in many cases *an impossible diagnosis*, although one may be repeatedly correct because so many cases of carditis are associated at that age with pericardial adhesion. In adolescents and adults it can be made with more confidence. It is distinguished from multiple serositis by the absence of any indications of a subacute process in the pericardium, pleurae, or peritoneum.

The prognosis. When there is every evidence of general external and internal adhesion the prognosis is bad; for the rest each case must be judged by the symptoms.

Treatment

There are no special indications for medicinal treatment other than those indicated under chronic valvular disease.

Bauer's operation of 'cardiolysis'. This surgeon showed at the German Congress in 1903 three patients in whom he had obtained improvement by freeing external pericardial adhesions. This procedure is still in the experimental stage. The indications are not easy to lay down, and the difficulties are manifest, but the hopelessness of the condition produced by severe external pericardial adhesion justifies a careful consideration of all measures that may give a gleam of hope. In childhood it must be remembered

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that the chest wall moves readily with the heart on account of its natural elasticity, and that a formidable operation may not improve upon this.

B. CHRONIC RHEUMATIC VALVULAR DISEASE

This section is concerned with the after-results of rheumatic valvular disease. The active condition has subsided, leaving behind it the healed valvular lesions.

This conception is a valuable one, for it enables us to picture disease of the heart, disentangled from its primary cause. Whatever that primary cause may be, whether rheumatism, tubercle, syphilis, or atheroma, the after-results, so far as the valves are concerned, produce much the same type of lesion, and there results incompetence or narrowing, or their combination, at one or more of the valvular orifices.

The after-results of rheumatic endocarditis can accordingly be used as the type of chronic valvular disease, and in future chapters it will be possible to consider those that are due to other primary causes, only in so far as they show special points, which in particular have bearing upon treatment.

1. Mitral Regurgitation.

This is the most frequent lesion. It does not, however, usually occur in a pure form, but is accompanied by some degree of contraction of the orifice, the necessary result of inflammatory thickening. This narrowing is as a rule insignificant, and not to be compared in its effect with the slowly progressive condition already described under chronic sclerosing endocarditis or true mitral stenosis, in which the lesion is often still active.

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Mitral regurgitation may arise in rheumatism from deformation of the flaps of the valve, or one or both may be tethered down by shortened chordae tendineae, and in addition, the contractility of the muscular ring may be damaged.

The mode of origin and early history of the condition have been already considered (p. 100), and the mechanical results explained (p. 45). There now remain for consideration the clinical features and course of the established disease.

Symptomatology. In childhood the compensated lesion gives rise to no symptoms except *shortness of breath and palpitation* upon over-exertion. When compensation fails *wasting* is an early symptom. The enlarging heart bulges forward the elastic chest wall, and the wide and diffuse impulse becomes one of the striking features.

Bronchitis and *broncho-pneumonia* are liable to occur, and gradually the aspect becomes cyanotic, and later still the skin icteric.

Dropsy at first is not a striking symptom, but in the last stages there may be severe general anasarca. As the condition progresses, breathlessness becomes more distressing, cough may be troublesome, and *pleural effusions* often increase the already existing embarrassment. *The liver* becomes enlarged and tender, and the urine scanty and albuminous. Among the terminal symptoms are *vomiting, diarrhoea*, and *restlessness*, with much *precordial distress*. This last stage may be painfully prolonged.

In adult life the symptoms differ in some details.

The compensated lesion, as in childhood, may give rise to no symptoms, even when an ordinary active life is led. The second stage, in which the first evidences of embarrassment commence to appear is more definite, for the adult

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recognizes his difficulties more rapidly. *Dyspnoea* and *cough* are early symptoms, and *dyspepsia* and *loss of mental power* and *emotional control* are also frequent. *Oedema* is a most important warning, and puffiness round the ankles at night may be the first symptom that brings the patient to the doctor seriously alarmed.

The third stage resembles in most respects the last stages in a child. The suffering is, however, greater, for *sleeplessness*, *pulmonary congestion*, *loathing of food*, and *tenderness and engorgement of the liver* are all more acutely realized. *Dropsy* is more extreme, and the helplessness of the patient greater. In the last stages the sallow hue of the skin, watery bile-stained conjunctivae, and cyanosis are so striking that at a glance one may recognize the nature of the case.

The physical signs will furnish some guide to the severity of the lesion in the individual case, and the following are some of the more important points for investigation:—

I. *The pulse.* This, when characteristic, is strikingly



FIG. 15. Irregular dicrotism. From a case of double mitral disease with poor compensation (T. Lewis).

irregular in force and frequency. The wave is moderate in size, easily compressible, and not felt between the beats.

With improvement it becomes more regular, with the reverse the irregularity increases, the wave becomes smaller,

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and many of the systoles may fail to produce a pulse at the wrist.



FIG. 16. Hyperdiastolic. From a case of mitral disease with fair compensation (T. Lewis).

II. *The heart.* 1. In childhood great deformity of the chest points to great enlargement of the heart and a severe lesion. Even in adults some degree of deformity may exist, for rheumatic heart disease usually begins in early life.

2. A systolic thrill is unusual, but palpation will, from the position and character of the impulse, give information as to the extent of the lesion and the relative predominance of hypertrophy and dilatation. An impulse which is feeble and tapping, and displaced far outward, points to a grave lesion.

3. The outline of the precordial dulness is more circular than usual, for it is enlarged to the right, left, and upwards. The greater the enlargement and the nearer circular the outline, the more severe, in general terms, is the lesion.

4. Interpretation of the auscultatory signs needs caution. No murmur is more variable in character than the systolic mitral.

(a) If it follows the first sound, is short, and but little conducted, the leakage is slight.

(b) A long murmur far conducted and accompanying the first sound points to a considerable lesion.

(c) A short, soft, whiffing murmur with a rapid, feeble heart and urgent symptoms means a very serious lesion.

Other features of this murmur are that it may vary with the irregular heart-beat, and be sometimes musical in character.

5. Attention must next be directed to the right side of the heart.

A heaving epigastric impulse points to a labouring right ventricle, a systolic tricuspid murmur to incompetence of that valve, and extensive dulness to the right of the sternum to marked dilatation of the right auricle. If these are present they mean that the mitral reflux has told severely upon the last line of defence.

Finally, the liver, veins in the neck, lungs, and subcutaneous tissues are investigated, and thus a very fairly accurate idea of the condition of the patient is obtained.

2. Mitral Stenosis

It was pointed out in Chapter vi, p. 105, that it was exceedingly difficult to tell when the active lesion ceases in this condition, but for practical purposes there is left for consideration the *final stage* of failure. So long as the lesion is thoroughly compensated the symptoms are almost in abeyance. Should, however, the condition have arisen in childhood—the earliest example of which in my experience has been at three years—growth may be stunted. The bright persistent flush on the face is also often present, and there is breathlessness on over-exertion. In this condition of compensation women have lived for years, becoming the mothers of considerable families, and to my knowledge such a patient has passed through nineteen confinements. Yet there are not unfrequently such warnings, as *epistaxis*, *haemoptysis*, or *infarction*, and in childbirth *fatal syncope* may

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occur or the limits of compensation be overstepped never to be recovered.

Symptomatology. Early evidence of failing compensation are *pain, dyspnoea, and palpitation*. The crimson flush becomes more purple, and there is *gradual loss of flesh*. *Chronic renal disease*, possibly also rheumatic in origin, may complicate the illness, and uraemic symptoms then appear.

Bronchitis is troublesome, and recurrent, and may be complicated by haemoptysis. The abdomen becomes much distended, partly on account of the great enlargement of the liver, and partly because *ascites* is more frequent in mitral stenosis than in regurgitation.

Again, *Infarction* occurs more frequently in mitral stenosis than in any other valvular affections except the malignant forms.

These patients have often a remarkable vitality, and may rally from repeated breakdowns. When death occurs it is generally from gradual cardiac failure complicated by pulmonary congestion and albuminuria. Sometimes an attack of palpitation proves fatal, and in rare cases death is sudden.

Physical signs. I. *The pulse* in compensated mitral stenosis is regular in force and frequency, and the wave is small and not easily compressible. When compensation fails it becomes irregular both in force and frequency.

II. *The heart.* The *Impulse* is displaced outwards, and there is generally a localized *presystolic thrill* which gradually disappears as the right heart fails. The *cardiac area* is enlarged, especially to the right, and is more circular in outline than normal. At the impulse there is audible a short presystolic murmur leading up to a short sharp first sound, and a faint second sound, or this latter may be absent.

The pulmonary second sound is accentuated and reduplicated at the base. With complete failure of compensation the presystolic murmur disappears, the accentuation of the second sound diminishes, and a tricuspid systolic murmur appears. The *veins in the neck* are distended and pulsating, and the liver is enlarged, tender, and sometimes also pulsates.

3. Aortic Regurgitation

This condition, when rheumatic in origin, differs very considerably from the other forms considered later, associated with degenerative arterial and cardiac lesions (vide Chapter ix, p. 144). The early age at which the lesion is produced generally permits of great and effectual hypertrophy by the left ventricle. Again, the mitral regurgitation so often associated with the aortic disease is in rheumatic cases frequently the result of *actual valvular damage* and not of relative incompetence.

Symptomatology. The compensated lesion may give rise to no symptoms, but there is generally *shortness of breath* upon exertion. The aspect is a contrast to that of mitral disease, for the face is generally *pallid*, particularly in these rheumatic cases. *Nose-bleeding* is a common occurrence, and *cerebral symptoms* are more often met with early in the illness than they are in mitral disease. Vertigo, sleeplessness, beating in the head, and *syncopal attacks* are among the more common evidences. *Pain*, too, is a frequent occurrence in aortic lesions. This may be no more than a sense of oppression behind the sternum at the base of the heart, or there may be severe paroxysms of angina, or, lastly, more or less continuous pain of considerable severity. The paroxysmal attacks, as in angina pectoris, are asso-

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ciated with gastric distension, belching, and flatulence. Muscular exertion excites them, but in advanced disease they may occur day or night irrespective of such exertion. In the late stages *cough and dyspnoea* may prove very troublesome, and there may also be *deranged mental conditions*, and sometimes insanity clouds the end of life. These mental cases are most difficult, and may become very unmanageable.

Aortic disease in childhood is usually a combination of organic mitral and aortic regurgitation. The clinical study of these cases necessitates a consideration of two main types: (1) one in which the aortic lesion is predominant, (2) the other in which it is of quite minor importance.

This division, too, is quite in accord with *post-mortem* investigation, for there are many occasions in which the aortic valves have been slightly damaged, and yet during life there has been no suspicion of aortic disease. Again, in other cases the lesion, though giving rise to a diastolic aortic murmur, has proved to be a very slight one, and the symptoms throughout have been entirely mitral.

In the first group in which the aortic lesion predominates there is well-marked pallor. Epistaxis, pain, breathlessness, nervousness, throbbing vessels and syncopal attacks are all symptoms found in childhood. The condition is one of much gravity at this age, and it may never really pass into the category of chronic valvular disease, death occurring from a fresh attack of rheumatism before the patient reaches adult age.

The second group, in which the mitral lesion is the prominent one, is more favourable, although the danger of another attack of rheumatism is ever present. In some the slight aortic lesion disappears, and this class, as regards the symptoms, can be classed under the mitral ones.

The Physical Signs. The physical signs of chronic aortic regurgitation are striking, and form some guide to the extent of the damage.

The particular points for investigation are :—

1. *The pulse.* The rate is increased, but the rhythm is usually regular, or there may be an occasional dropped beat. The wave is large, sudden, ill-sustained, and may give in diastole a curious sensation to the finger as of a back current of blood in the vessel. The vessel is visible, and the arteries

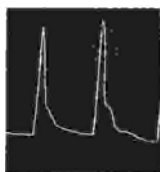


FIG. 17. Extreme aortic regurgitation, with little or no sclerosis of vessels, rheumatic in origin (T. Lewis).

in the neck, at the temples, and in the fingers beat in the same abrupt fashion. This is termed the *water-hammer* pulse. If the wrist is put to the ear the wave is audible. In some cases there is a marked delay between the heart-beat and pulse-beat.

The collapsing nature is best realized by grasping the forearm lightly with the hand and raising it above the level of the heart.

The more collapsing the pulse the more severe the actual aortic lesion. The difference between the systolic and mean blood-pressure in aortic regurgitation is necessarily great. In many cases there is *capillary pulsation* which can be brought out by smartly stroking the skin over the forehead or pressing upon the nails. Occasionally there

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is visible arterial pulsation in the retina, and sometimes a pulsation in the veins on the back of the hand.

2. *The heart.* In these rheumatic cases there may be enormous hypertrophy, and the impulse be displaced downward and outward to the sixth space or seventh rib. The *heaving impulse* is a good measure of the power of the ventricle.

The outline of the *cardiac dulness* is more elliptical than in health on account of the lengthening of the left ventricle.

A large area of precordial dulness points to a considerable lesion.

The diastolic murmur with its various points of maximum intensity has been already described. In these cases it is usually soft, and may follow the second sound or obliterate it. Upon auscultation in the neck over the carotid, the diastolic murmur can generally be detected, and its relation to the aortic second sound is best determined in this way. The lesion is greater when the sound is obliterated than when the murmur follows it. In some instances a *presystolic* murmur is heard at the apex and a question arises as to its correct interpretation. Sometimes this is the result of mitral stenosis and in other cases the mitral valve is healthy. In settling the diagnosis, attention must be directed to other evidence of mitral stenosis. The large pulse of aortic incompetence would be modified by organic mitral stenosis which is associated with a small pulse-wave. The first sound at the impulse would be abrupt, the right side of the heart hypertrophied and the cardiac outline for this reason more circular than is generally the case in pure aortic regurgitation.

The diastolic murmur of aortic regurgitation, it should be remembered, may *disappear* when there is acute failure of

the left ventricle. This is a point of some prognostic importance.

Lastly, attention must be directed to the condition of the *right side* of the heart, for if there is mitral regurgitation, whether from organic damage or relative incompetence, it is important to estimate the degree. This may be done as described under mitral regurgitation.

4. Aortic Stenosis

This lesion resembles the preceding in occurring in two different types. One, the *cardiac*, which is well represented by its most frequent cause, rheumatism; the other, the *arterial*, to be considered later (p. 147). In the opinion of some writers it is not expedient to draw a distinction between a mere thickening of the aortic valves or valve-ring and a stenosis of the opening, in that these are but degrees of the same condition. Clinically there appears to me to be a practical difference between a true stenosis and a thickening of the ring or the valves from which may result a double murmur. This thickening is repeatedly met with in cases which in their course are examples of aortic regurgitation and show all its peculiarities and dangers. The stenosis is, in such, negligible. In true aortic stenosis, on the other hand, the regurgitation is insignificant. This lesion is certainly less common than the preceding. The early stages of its development have already been considered (p. 108).

The degree of stenosis varies considerably, the thickened valves may be joined together leaving only a small central aperture or may only be crumpled and deformed, or again, they may be deformed and adherent, and, in addition, the ring of the valve much thickened.

Symptomatology. The symptoms are long delayed.

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Vertigo, pain over the heart, and dyspnoea, are the most frequent of them. Later there is wasting.

The Physical Signs. I. *The pulse* is slow, the wave is small and long, and the artery felt between the beats. An *anacrotic notch* may occur in the sphygmographic tracing,



FIG. 12. The anacrotic pulse. From a case of aortic stenosis (T. Lewis).

but this, though suggestive of aortic disease, is not pathognomonic. The character of the pulse is a very important factor in the diagnosis of aortic stenosis. It is so different from the pulse of aortic regurgitation that it is comparatively an easy matter, when both bruits are present at the aortic cartilage, to decide which of the two lesions is the important one.

II. *The heart* shows the degree of stenosis in the *hypertrophy* of the *left ventricle*, which in this condition may raise the entire precordial region with a measured, heaving impulse. Again, the contrast between this powerful heart and the small pulse is a striking feature of aortic stenosis.

The cardiac outline tends to the elliptical form. *The murmur* is usually harsh and vibrant, sometimes soft and blowing, and when the stenosis is considerable and the heart powerful there is a well-marked systolic thrill conducted upward into the vessels of the neck.

The aortic second sound is usually diminished in intensity. Lastly, should mitral incompetence supervene, attention, as in aortic regurgitation, must be given to the condition of

the right side of the heart. It will be found that when the heart fails the murmurs become less harsh and the systolic thrill may disappear.

5. Tricuspid Regurgitation

In acute rheumatism *tricuspid regurgitation* is generally the result of relative incompetence of the valves, produced by mitral incompetence or stenosis, or by acute dilatation of the entire heart. The valve is nevertheless damaged in a certain number of cases by rheumatic endocarditis and my experience coincides with that of Newton Pitt, who states that the dominant cause of such vegetations is a severe rheumatic process. When these vegetations are found on the tricuspid valve there is almost invariably considerable mitral and sometimes aortic disease as well.

The disease is generally not sufficiently extensive to produce a detectable tricuspid regurgitation or one of any clinical importance.

Symptomatology. The symptoms of tricuspid regurgitation in rheumatism are, then, almost invariably the result of relative incompetence.

The most important are *dyspnoea*, *cyanosis*, and *dropsy*. The *oedema* commences in the most dependent parts and spreads upwards.

Insomnia and *horrible dreams* are frequent. Effusions into the pleurae, albuminuria, congestion of the bases of the lungs, and bronchitis are very liable to complicate the condition.

Physical Signs. I. *The pulse* in some cases is small, easily compressible, and irregular.

II. *The heart.* The right ventricle impulse is often forcible and heaving.

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The *cardiac* dulness is increased to the right by the dilatation of the right auricle, and in some, but by no means in all cases, there is a systolic tricuspid murmur. The *pulmonary second sound* at the base is weak. The *veins* in the neck may be obviously pulsating. The *liver* is enlarged, tender, and sometimes there is true pulsation.

6. Tricuspid Stenosis

Rheumatism is the most frequent cause of this rare lesion. It is almost invariably associated with *mitral stenosis*, and as Newton Pitt has pointed out, the aortic valves are generally damaged also. In one of the three cases that have come under my own observation, the aortic orifice admitted the tip of the finger, the mitral the same, and the tricuspid the tips of two fingers. All three were examples of inveterate cardiac rheumatism in adults.

Symptoms. The symptoms come on gradually and are usually *dyspnoea*, *oedema*, *cyanosis* and *albuminuria*.

The Physical Signs. The physical signs that point to the diagnosis are the occurrence of a *short, sharp, first sound* in the tricuspid area with sometimes a *presystolic murmur*. In two of the above three cases there was a definite tricuspid, presystolic murmur. In many cases, however, no presystolic bruit has occurred.

Mackenzie found in cases of tricuspid stenosis that when a pulsating liver was present, the pulsation was auricular and not ventricular in time.

7. Pulmonary Stenosis and Regurgitation

These lesions practically never result from acquired rheumatic endocarditis of the valve. In advanced mitral stenosis there is sometimes a relative incompetence of the

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pulmonary valve, and in all probability some of the cases of congenital pulmonary stenosis (q. v.) are due to foetal rheumatism.

C. CHRONIC RHEUMATIC DILATATION

This is more frequent than is generally recognized and constitutes an important clinical group met with both in childhood and adult life. The condition of the heart resembles that which is sometimes left after attacks of influenza or diphtheria.

Symptomatology. The symptoms are *nervousness, anaemia, general debility, syncopeal attacks, and breathlessness* upon slight exertion.

The Physical Signs. *The pulse* is rapid, easily excited and often irregular. The impulse of the *heart* is displaced outward and is feeble and diffuse. The cardiac dulness is increased and the sounds are short and feeble. There is no evidence of an adherent pericardium and often no cardiac bruit.

These are intractable cases, and help to swell the group of irritable and weak hearts met with in adult life.

CHAPTER IX

CHRONIC NON-RHEUMATIC VALVULAR DISEASE AND THE GENERAL PROGNOSIS AND TREAT- MENT OF CHRONIC VALVULAR DISEASE

Degenerative aortic lesions—Aetiology—Aortic regurgitation and stenosis—Their characteristics—Degenerative mitral lesions—General prognosis of chronic valvular disease—Rupture of valves and other cardiac injuries—Treatment of chronic valvular disease—General principles—General management—Failure of compensation—In mitral regurgitation—In mitral stenosis—In aortic regurgitation—In aortic stenosis.

CHRONIC valvular lesions may result from infections other than the rheumatic. Scarlet fever is the most important. Diphtheria, influenza and measles are occasionally immediate antecedents. Syphilis is yet another cause and in rare cases tuberculosis and gout. In acute rheumatoid arthritis endocarditis has been frequently recorded, and it may be that some of the causes of malignant endocarditis may exceptionally produce the simple lesion and then chronic valvular disease, but such causes are more liable to produce the fatal malignant types. When chronic valvular disease does follow on endocarditis, it may be exceedingly difficult to ascertain the original cause, because all chronic valvular lesions, whatever their origin, show a great clinical resemblance.

Nevertheless, there is a generally recognized group of cases which are the result of degenerative changes in the valves. The exact causation of these changes may be, and

is, obscure, but the clinical characters of the valvular lesions are of much importance.

I. DEGENERATIVE AORTIC LESIONS

Prominent among this group are the degenerative aortic lesions which are almost invariably accompanied by similar changes in the *first part of the aorta*.

In some instances the disease has actually commenced in the artery and extended to the aortic valve ; in others there has been long-standing chronic diseases of the valve and this has later implicated the artery.

A distinction has been drawn between the *cardiac* and the *arterial types* of aortic disease, but this is not absolute, for they overlap one another ; nevertheless it has considerable practical value.

The most frequent cause of the cardiac type of aortic disease is undoubtedly *rheumatism*.

Aetiology. The arterial type, on the other hand, is associated with *gout, syphilis, atheroma, and general arterio-sclerosis*. This form is met with more frequently in the elderly, the cardiac is more frequent in young subjects. *Males* are more often sufferers than females. *Muscular overstrain* and *persistent high blood-pressure* are also factors in the causation.

The morbid anatomy. The striking features in these cases are the *arterial changes*. The peripheral arteries may show much arterio-sclerotic change, and the first part of the aorta most extensive atheroma. Another important point is the implication of the openings of the *coronary vessels* by the atheromatous process. The aortic valve itself is rigid and often calcified. In some instances the bases of the cusps are greatly thickened and fused together, thus producing extreme stenosis.

(a) Aortic Regurgitation

Symptomatology. The onset is very insidious. *Giddiness, shortness of breath, sleeplessness*, and above all, *cardiac pain* are the most prominent. In this type of aortic disease there is more often *severe angina* than in the cardiac form, because of the involvement of the aorta and the coronary arteries. In some cases there is the *pallid, anxious face* of aortic regurgitation but in others the *colour may be high* and the face round. There may be many other symptoms which are referable to degenerative lesions in other organs rather than in the heart. Such are *mental changes* from sclerosis of the cerebral arteries, *dyspnoea* from pulmonary emphysema, and *asthmatic attacks*, the result of chronic renal disease.

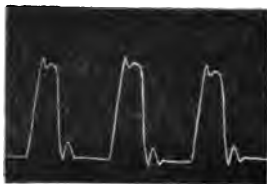


FIG. 18. Extreme aortic regurgitation, with very pronounced sclerosis of arteries syphilitic in origin (T. Lewis).

The physical signs. (1) *The pulse* is not so characteristic as in the cardiac type, for the rigid arterial wall tends to deceive the finger by masking the collapsing nature of the wave. Some definite degree of stenosis is also more often met with in these cases and this again modifies the pulse. If these peculiarities are not present the pulse will be collapsing and typical of aortic regurgitation.

(2) The physical examination of the *heart* may show very considerable variation in the signs in different cases.

In some, and these are highly dangerous, there is but little hypertrophy and the diastolic murmur may be only detected by greatest care, being low-pitched and audible over only a very circumscribed area. In other cases there is great enlargement of the cardiac dulness, the result of dilatation rather than hypertrophy. Lastly, there may be, as in the cardiac type, great hypertrophy and dilatation of the left ventricle. The diastolic murmur, which is usually associated with a systolic one also, is in these cases loud and long, and audible down either side of the sternum.

A *pure aortic lesion* is more often met with in the degenerative cases than in those of the rheumatic type, for the mitral valve is not so often attacked by this process as by the rheumatic.

Relative mitral incompetence may, however, result as a secondary process. *General dilatation of the arch of the aorta* may also accompany the aortic regurgitation and give rise to percussion, dulness over the manubrium sterni and to a well-marked shadow upon the fluorescent screen.

The course of these lesions is very uncertain, and, as would be expected from their degenerative origin, not hopeful. *Angina pectoris, sudden syncope or renal complications* may cause rapid death. A more *gradual cardiac failure* is, however, the rule.

(b) Aortic Stenosis

The morbid anatomy of these cases is similar to that found in the regurgitant lesions, though it must be added that the stenosis may be produced by a narrowing of the left ventricle itself immediately below the level of the valve, which is little if at all affected.

Symptomatology. As a rule there are no symptoms when the stenosis is slight, but if it reaches a high degree, *vertigo, insomnia, breathlessness and vague precordial pain* are

complained of. It is remarkable what an extreme degree of stenosis is compatible with existence, but in such cases most dangerous symptoms may arise, such as *epileptiform*, *apoplectiform*, or *syncopal attacks*. These are associated with a slow pulse and are described under the Stokes-Adams syndrome, p. 249.

The Physical signs. The *pulse* is small in volume, infrequent, and the wave prolonged, deliberate, and not easily compressible. There may be an anacrotic pulse. In this type the arterial wall is often thickened and tortuous.

The *heart* is enlarged, and the left ventricle heaving, but this feature may be masked by the occurrence of pulmonary emphysema.

The systolic aortic murmur is often loud and harsh, conducted up into the vessels of the neck, and there is usually a systolic thrill.

In some cases the aortic second sound is louder than usual, in others the intensity is much diminished.

The diagnosis of aortic stenosis is more difficult than that of aortic regurgitation on account of the variety of basal systolic murmurs.

Four of these must be remembered in particular :—

1. The systolic murmur produced by an aneurysm.
2. The systolic murmur produced by dilatation of the aorta resulting from aortitis or atheroma, the aortic valve being itself intact.
3. Functional or hæmic murmurs.
4. Organic pulmonary murmurs.

The diagnostic signs of *Aneurysm*, *atheroma* of the aorta, and *aortitis* are considered in Chapter xiv.

Functional murmurs are less harsh, do not produce a systolic thrill, and are not conducted into the vessels of the neck. None of the characteristics of aortic stenosis are

discoverable in the pulse, and the left ventricle is not hypertrophied.

Organic pulmonary murmurs are almost invariably congenital, they are more pronounced to the left of the sternum, and are not conducted upward into the neck. If there is pulmonary stenosis the right and not the left ventricle will be hypertrophied.

The course of this form of aortic stenosis is very slow and gradual, and this lesion is sometimes met with in patients of advanced age.

II. DEGENERATIVE MITRAL LESIONS

These lesions associated with degenerative valvular changes, resemble the rheumatic form of chronic mitral disease, except that they are more usually met with after middle age and may be complicated by *degenerative changes* in the myocardium. It is these myocardial changes which are of interest and are considered in Chapter xiii.

THE GENERAL PROGNOSIS OF CHRONIC VALVULAR LESIONS

1. **Mitral regurgitation** is the least harmful of the endocardial lesions, and is compatible with good health lasting for many years. An *exception* must be made to some cases of mitral regurgitation of rheumatic origin in early life in which there is a great dilatation of the mitral ring. In these cases there is a loud, long, systolic murmur, with a greatly enlarged heart, to which the pericardium may be adherent. The symptoms in such cases are always severe. Mitral regurgitation due to atheroma of the valve in the elderly may never give rise to any symptom of importance.

Under the physical signs of mitral incompetence some of the details in the making of a prognosis in particular cases are indicated.

2. **Mitral stenosis** is a more serious lesion. Pulmonary complications are frequent and embolism may suddenly produce serious or even fatal lesions. In early life the development of the patient is interfered with and there is a greater tendency for the active disease to persist. The stages given in Chapter iv are useful guides for arriving at a prognosis in the individual case.

Where there has been a failure of compensation, the prognosis will depend upon the *exciting cause* of the failure, the *number of previous breakdowns*, and the *reaction* of the *patient to treatment*.

The question sometimes arises as to whether a woman with a mitral stenosis should marry. The dangers should be explained, but further than this the physician can hardly go if the lesion is well compensated and health good. Many women have borne families under these circumstances, and although these patients frequently die under forty, and rheumatism is also strongly hereditary, we cannot deny that healthy and clever children have resulted from such marriages. In any case the decision is generally taken out of the doctors' hands.

Pregnancy and *labour* undoubtedly increase the risk attendant upon this lesion.

According to the Broadbents, in any case the average length of life in males suffering from mitral stenosis is thirty-three years, and of females thirty-eight.

3. **Aortic regurgitation** is less serious when the result of endocarditis than when it is the outcome of degenerative changes. A well-compensated lesion is compatible with years of useful life. When the lesion is the

result of degenerative changes, and particularly when there has been previous high blood-pressure, the prognosis is uncertain and the length of life is generally much curtailed. Syncopal and anginal attacks are dangerous signs.

Much depends upon the life and habits of the patient, and a good deal can be learnt from a careful study of the reserve power of the heart. If this is slight there will be a history of heart trouble from slight exertion, emotion, or mental strain, and the prognosis must be correspondingly guarded. The indications to be learnt in individual cases from the physical signs are given under the symptomatology, p. 137. In some cases, for example syphilitic ones, the myocardium may be damaged, and the coronary vessels diseased; evidence of myocardial weakness is then a grave indication of danger.

Sir W. Broadbent points out that in some cases the supervention of stenosis may make the prognosis more hopeful.

4. **Aortic stenosis** is looked upon by some as the least serious valvular affection, but it is probably to be placed second to mitral regurgitation, partly because it is often accompanied by some aortic regurgitation, and partly because there may be degenerative lesions of the myocardium the result of concomitant coronary disease. If the stenosis is practically pure, its slow onset and the sure response of the left ventricle to the additional strain make it a lesion compatible with many years of usefulness.

5. **Combined mitral and aortic disease.** Under twelve years of age the combination of mitral and aortic disease is a very serious one, for there is a great likelihood of further attacks of cardiac rheumatism.

III. RUPTURES OF VALVES AND CARDIAC INJURIES

These include damage to the endocardium, myocardium, and pericardium ; but the lesions of the most importance to the physician are *ruptures of the cardiac valves*.

The aortic valve suffers the most frequently, though the mitral may also be damaged.

The cause of rupture may be external violence, as from a crush of the chest, or a sudden overstrain on the part of the patient.

Sudden over-exertion may rupture a healthy aortic valve, but it is far more usual for the segments to be to some extent diseased. In malignant endocarditis a rupture may result from the mere strain of the blood-pressure upon the ulcerated valve, and this can only be looked upon as a complication of the acute process and not as an injury. It is when a middle-aged man with some degree of atheroma of the aortic valve tears a cusp by some imprudent exertion that the most characteristic examples of this condition occur.

Symptomatology. In the great majority of cases there is, at the time of rupture, *sudden pain* in the chest and *breathlessness*, or even *fatal syncope*. The patient usually *stands still* or leans momentarily for support, and he may *vomit*. After a short rest he may recover sufficiently to continue to exert himself again, and then find that he is *breathless* and that his heart is *acting tumultuously*. It should be borne in mind that a rupture may occur without any noticeable *degree of pain* if the valve is already diseased. This is a point of considerable medico-legal importance in reference to the Workmen's Compensation Act.

The physical signs are those of the particular valvular lesion. In the most common, the aortic, there are the *physical signs of aortic regurgitation*, and the two loudest

murmurs I have ever heard have been the result of ruptured valves.

Prognosis. As is to be expected, the lesion is a very serious one, particularly if the patient is middle-aged, for there is a sudden demand upon the reserve power of the left ventricle which it only meets very imperfectly, and dilatation far outstrips hypertrophy ; and although a prolonged rest may cause a disappearance of symptoms, ordinary exertion soon brings into evidence again the grave injury to the heart. Life may be prolonged for months, but the patient is an invalid, and in many cases death follows within twelve months from complete cardiac asthenia.

Injuries to the myocardium and pericardium by external violence are chiefly of surgical importance. Death is often sudden, or if not, there is great shock ; the pulse at the wrist fails and the action of the heart is feeble and irregular.

The most important medical cases are those in which there has been severe damage to the chest, the valves have not been injured, but apparently the myocardium has been bruised by the injury. When the patient recovers from the shock and begins to get about again, he complains of pericardial pain and breathlessness. This condition may remain obstinate to treatment and be the starting-point of a complete breakdown. Lastly, the shock of a heavy fall may, in aged people, produce a rapid or gradual cardiac failure.

IV. THE TREATMENT OF CHRONIC VALVULAR DISEASES

All forms of chronic valvular diseases, including the rheumatic, are considered ; for whatever the cause of the original valvular lesion, the chief indications are the same ;

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where there are special points, these will be brought into prominence.

I. General Principles

The first is to consider the individual, and not to be content with treating the heart alone.

The second is not to interfere with the heart when there is a well-compensated lesion.

The third is to intervene as soon as possible when there are signs of failing compensation, and to look upon the frequent and terrible breakdowns so often met with among the poor as disasters comparable to such surgical calamities as protracted cases of unreduced strangulation and other forms of intestinal obstruction.

II. General Management.

(a) *When compensation is effectual.*

Many of the rheumatic patients are young, and questions of *education* and *amusement* must be considered. A firm stand should be made against their parents permitting them to become mental invalids, and an ordinary education should be advised. *Exercise* is equally important, and the character and amount to be allowed can usually be ascertained if the physician is supported by reasonably good observers who are in charge of the child.

Exercise can be usefully divided into these grades:—

1. Walking at a measured pace along the flat, and up moderate inclines. Systematic muscular movements.
2. More active physical exertion, such as swimming, riding, cycling, golf, and dancing.
3. Active sports, such as tennis, cricket, racquets, boxing, football.
4. Racing.

The active sports vary much in their severity, and curious mistakes are made by those who are not practically acquainted with them. Racquets and boxing, for example, are allowed when cricket is debarred, yet racquets is a very fast game played in a closed court, and boxing is very severe exertion.

All racing is out of the question, and football is too great an exertion on account of the sudden severe muscular strain it involves. Many children and adolescents with slight valvular lesions can take part in most of the other forms of amusement, short of over-fatigue. In every case, however, undue breathlessness and pallor must mean that there is danger.

I think it a mistake to send a boy to a large public school when the heart is only capable of the mildest forms of exertion, and particularly is this the case when he is keen and sensitive.

The parents of rheumatic children should be told the importance of sore throat, fleeting pains, nervousness, and wasting as indications of renewed activity of the disease.

I have followed many children with valvular disease from childhood through puberty to adolescence, and have not found that the great development which occurs during those years has any great effect upon the heart. If there should be advanced mitral stenosis, it is the general physical development rather than the heart that suffers, and even when there is apparently a very considerable mitral or aortic regurgitant lesion growth and development may continue with remarkable vigour.

In *elderly persons* with valvular disease the question of exercise is a very different one. Over-exertion may do irreparable harm to them, and although steady, moderate, daily exercise is indispensable, the old sportsman should

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realize that discretion is better now than valour, and in direct contrast to the child or adolescent, gradually curtail his activity.

Clothing should be warm and the extremities protected.

The bowels should be regulated, a point of first importance.

The diet in the young should be ample and plain, and the nitrogenous food should not be curtailed during the time of active growth and development.

In *chronic valvular disease* later in life special attention must be paid to the blood-pressure, and if this is high, meat should be restricted. Peculiarities of digestion will need to be carefully studied, and sufficient time should be given to the meals.

Stimulants are unnecessary in early life, but adults who are accustomed to some wine or beer with their meals are permitted a good claret or hock, diluted spirit, or the lighter beers. *Smoking* must be strictly moderate.

The counsel of perfection would be no alcohol and no tobacco.

The *best climate* is seldom within our reach. A dry gravel soil, southerly aspect, and bracing sunny situation are the qualities to be desired.

In cases of organic heart disease the equable life with all emotions under strict control is the ideal one, and this ideal can at least be laid before the patient.

(b) *The treatment of failure of compensation.*

1. *Rest.* This is of the greatest value, and most especially so when the cause for the breakdown is to be traced to some definite over-fatigue, over-strain, or accident. In rheumatic cases it will be remembered that the most frequent

cause for a breakdown is a rheumatic attack ; this too will equally require rest.

Rest is least satisfactory in cases that have broken down gradually without any evident reason, and in which there are restlessness and sleeplessness when the patient is kept confined. Nevertheless, in these cases one good attempt should be made to test its value.

When in the last stages of heart failure rest has been tried without effect, it is impossible to formulate rules. The management then depends upon the practitioner's knowledge of his patient and his own common sense.

2. Diet. The digestion is usually now at fault, and in accord with the severity of the case, a restricted diet or even an absolute milk diet is advised.

For an example of a restricted diet vide Appendix, diet 1.

When there is much *dropsy* the fluids should be restricted. A very rigid diet of this type is also given in the Appendix, diet 2. Recent researches favour also the view that the chlorides should be much restricted. Vide Cardiac dropsy, Chapters iv, p. 74, and xiii, p. 231.

Stimulants are required in the later stages, and are then given as medicines in regular doses at regular intervals, but at first, if the patient is accustomed to their use with the meals, some wine or diluted spirit can be taken then. Sir W. Broadbent considers six ounces of brandy in the twenty-four hours the limit in bad cases.

There are two great dangers in the use of alcohol :—

1. The risk of subsequent dram-drinking.

2. An abuse of the remedy, by permitting too great a quantity, and keeping up its use too long after the urgency has passed.

The invalid's room must be freely ventilated, and should be, if possible, bright and sunny.

3. **Medicinal treatment.** This will depend upon the age of the patient, the cause of the breakdown, the nature of the particular valvular lesion, its severity, and its original cause.

A. Mitral Regurgitation

There are two fundamental principles :—

1. To diminish the resistance to the heart's force.
2. To increase the strength of the heart.

These two principles are complicated by the occurrence of *secondary symptoms*, which may be so prominent as to demand immediate attention, or may at least interfere with the successful employment of the cardinal measures. Sleeplessness, vomiting, and serous effusions are among those which will at once occur to the reader.

There are many uncomplicated cases which, if they are taken early, will at once respond to rest, purgation, careful dieting, and a steady course of digitalis, followed very possibly by a complete change of scene and climate.

In other cases the secondary symptoms may need much treatment before any advance can be made.

First as to emergencies occurring in the adult.

The most important is over-distension of the right side of the heart. The patient is livid and dyspnoeic, the pulse at the wrist small and easily compressible, the right ventricle impulse forcible, and the dulness to the right of the sternum much increased.

It is possible that rest, warmth, an immediate injection of 5 minims of the hypodermic solution of *strychnine*, and three grains of *calomel*, followed in four hours by a *seidlitz powder*, will suffice to adjust the balance again. In other cases some form of *blood-letting* will be imperative. Venesection to twelve ounces or eight leeches applied to the liver will then

afford great relief. This prompt treatment must be at once followed by free stimulation of the heart, to complete the restoration. *Digitalis* takes time to act, and three days at least are necessary to judge of its result, accordingly it is advisable to combine with it *strychnine*, which acts more rapidly as a cardiac tonic, although not so effectually.

If the peripheral resistance is high, *nitro-glycerine* should be added to the mixture, or *digitalis* and *iodide* combined, and *strychnine* given hypodermically. *Stimulants* should be given freely, and these remedies should be pressed for at least twenty-four hours.

Another emergency is extreme dyspnoea, produced not so much by right heart failure as by a pleural effusion, particularly when it is on the right side. No time should be lost in *removing the fluid*.

Digitalis is the most valuable remedy in mitral incompetence ; it may be given as the powdered leaf in pill or cachet, dose 1-3 grains.

As the tincture in doses from 5 to 30 minims.

As the infusion in doses from $\mathfrak{z}\text{ij}$ - $\mathfrak{z}\text{i}$.

As digitalinum pulverizatum purum Germanicum, in doses of $\frac{1}{160}$ - $\frac{1}{80}$ of a grain, prescribed in tablets of $\frac{1}{160}$ th grain.

Since in all matters of treatment opinions differ, such indications will be given as have appealed to the author as useful. *Digitalis* steadies the heart, stimulating the cardiac muscle and causing in addition arterial constriction. Diastole is prolonged and free diuresis produced. The best results are to be expected when the blood-pressure is low. Unfortunately it may also produce nausea, vomiting, and diarrhoea.

Its hypodermic use is especially indicated when the digestion is easily upset. *Vaso-dilators* (10) should be combined when the peripheral resistance is high, and

caffeine (9) may be valuable in combination when the renal secretion is very scanty, or a tendency to asthmatic attacks is present. It is, I think, a mistake to stop digitalis as a matter of routine after a few days' use in order to anticipate symptoms of poisoning, and I prefer to press the drug at first and then to diminish the dose gradually.

Before a course of digitalis, the *bowels* should be thoroughly opened by a *mercurial* combined or not with *compound jalap powder*, or *colocynth*. For the delicate one grain of calomel may be sufficient, or equal parts of the blue and compound rhubarb pills to make a pill of 5 grains, if followed by a saline; the more drastic combination with $\frac{1}{2}$ a drachm of the compound jalap powder is suited to the robust cases with oedema, a high blood-pressure, and congested liver.

In cases where there is suspected myocardial weakness it is important to insist on the use of the bed-pan. When the bowels have been thoroughly opened, *mild aperients* or a morning saline if effectual are preferable.

It is in mitral regurgitation that we expect to find great service from digitalis, but its beneficial effects may be retarded by obstinate insomnia, or insomnia and precordial pain and distress.

Hypnotics may be required, and of these trional, veronal, paraldehyde, and chloralamide are among the safest. *Trional* can be prescribed in cachets, dose 10-30 grains; *veronal* may be combined with bromide, and is suited to the more weakly and nervous cases. Unpleasant consequences may follow large doses, and this drug by no means suits all. Five grains of veronal and ten of bromide of potassium may nevertheless produce a refreshing sleep when other remedies fail.

Paraldehyde is a nauseous drug to take, but can be prescribed in cachets, of 20-30 or 60 minims, or in solution (4): it is often best given in divided doses, e.g. 30 minims early

in the evening followed in two hours by another dose of the same size.

Chloralamide can be given in doses of 20 or 30 grains.

When there is pain and mental suffering, *opium* is indicated. The best preparations should be used, and among these *nepenthe*, which is of the strength of tincture of opium, is one of the most satisfactory. In cases of this kind it may be combined in ten or fifteen minim doses with *digitalis*, and given not at night only but during the day, every four or six hours. Undoubtedly opium upsets some patients, and when there is great respiratory distress is somewhat dangerous; but excepting these drawbacks there is no substitute for it in some of the most difficult cases of insomnia and pain. *Morphia* may be required in aggravated cases. The dose should be as small as possible, one-eighth of a grain may be sufficient. The great objection to *morphia* in these cases is the shock that follows the sudden injection of the drug into the system; this precedes the soothing action of the remedy. All opium preparations should be commenced cautiously, for it is impossible to guard against an idiosyncrasy in their action.

Again, *digitalis* may fail because there is extensive *dropsy*, embarrassing all the functions, and in such cases the oedema can be relieved by draining the legs by Southey's tubes or multiple puncture. Very special precautions must be taken to prevent any septic infection. Whether tubes or multiple puncture be used depends greatly upon the patient; if he is troublesome the tubes will probably not be tolerated (vide Chapter xiii, p. 223). Indigestion must be treated on general principles, and flatulence combated by carminatives and gastric antiseptics (11).

A troublesome harassing cough may be a source of much suffering. Demulcent drinks are useful, and a linctus con-

taining *heroin* or *morphia* (12) may afford great relief when the paroxysms are severe. On the other hand, a hard, dry, ineffectual cough may indicate an effusion into the chest which should be tapped.

When *digitalis* is successful, there is free diuresis; the pulse steadies, the heart slows, the area of cardiac dulness diminishes, and the impulse improves in tone.

With improvement the dose is diminished, the diet increased, and the alcohol gradually withdrawn. *Massage* will now assist in dispersing the dropsy, and *passive movements* will accustom the heart to more vigorous exercise. A cardiac tonic should be persevered with until the compensation is established for ordinary life.

In spite of all precautions *digitalis* may fail, then it is much to be feared that the case has reached that last stage when nothing seems to be of service. It is again a frequent experience to find improvement up to a point, and then a standstill is reached which sometimes represents the patient's limit. It must be admitted that then in some cases the *removal of all medicines* may be followed by improvement.

Where *digitalis* fails *strophanthus* may succeed; but it is not so satisfactory, although the direct action upon the cardiac muscle is more pronounced, and the constricting effect upon the arterioles less. Ten minims of the tincture may be given every four hours. The combinations of strychnine and *digitalis*, and caffeine and *digitalis*, have already been mentioned. For caffeine, *diuretin* in 15-grain doses may be substituted when there is much dropsy, and *apocynum cannabinum*, in 15-minim doses of the tincture, has been praised for its effect upon cardiac dropsy.

B. Mitral Stenosis

The general management of the compensated lesion presents no unusual features. Particular warning must be given to the patient not to overstrain the heart. An overstrain may mean the starting-point of a complete breakdown or of frequent attacks of palpitation. The diet should not be large but sufficient; a great bulk of solid or fluid consumed habitually raises the blood-pressure, and throws undue strain on the weak left ventricle. The pulmonary circulation, which is carried on under difficulties, must be carefully protected from chill.

The special difficulties when compensation fails are the *failure of digitalis*, the frequency of troublesome palpitation and dyspnoea, and the occurrence of infarction or of chronic renal disease.

All authorities seem agreed that digitalis is not well borne, and often does more harm than good. It may be usefully employed in these cases when there is acute dilatation with much tricuspid regurgitation. The bowels must be kept thoroughly open, and the drug not administered for more than a few days at a time, its place being taken by other cardiac tonics in the intervals.

There are the same indications for venesection, leeching, purgatives, and tapping of effusions as in mitral regurgitation.

Strychnine alone, or *ammonium carbonate* (13), and *nuxvomica*, with or without *arsenic*, are useful tonics, and in many cases a 3-grain dose of *iodide of potassium* or a minim of *nitro-glycerine* solution, are indicated when the blood-pressure is high. These may be used persistently for weeks.

Palpitation is sometimes the last phase in mitral stenosis,

and all treatment may prove unavailing, but before this, there may have occurred attacks which have passed off under careful management.

A *belladonna* plaster should be applied to the precordium. Acidity and flatulence may be treated by *alkalies* and *carminatives*, for example, drachm doses of compound spirits of ether. Tea should be stopped, and alcohol if it should excite the heart's action. *Digitalis* often fails, but may be tried, in a first attack, in 10-minim doses of the tincture combined with 15 grains of *ammonium bromide*. If there is a large mental element, *valerianate of ammonia* may be combined with *ammonium bromide* and *camphor* (14). If the heart is feeble, *strychnine* may be successful, but should be abandoned if it only increases the excitement. Closely allied to this palpitation is paroxysmal dyspnoea, for which Lees finds much good from the employment of *atropine* in a full dose of $\frac{1}{25}$ th of a grain (4 minims) injected subcutaneously. If the patient can be watched, an *ice-bag* applied to the precordium is worthy of trial.

Lastly, if restlessness and mental anguish are prominent features, *opium* combined with *bromides* is indicated.

The pain of infarctions. is best combated with *morphia* or other *opiates*, due attention being paid to the condition of the kidneys and degree of cyanosis.

C. Aortic Regurgitation

In the treatment of this affection the difference between the cardiac and arterial types must be kept in view.

1. **The cardiac type.** In no form of valvular disease is *rest* during the healing of the valve more important than in this. Six months is not too long.

When compensation is affected, except for occasional tonics

no drugs are needed, but occasional periods of rest are most useful for the aortic regurgitation of young people. Anaemia is a symptom to be watched carefully.

The general indications are the same as in mitral cases, but the lesion, when rheumatic, usually means a more severe type of infection, and exercise must be more curtailed.

Failing compensation indicates rest, and such cardiac tonics as *iron*, *arsenic*, and *strychnine* (15). *Digitalis* is indicated in aortic cases when there are mitral symptoms, but needs special care in childhood, for then the pulse may rapidly become very slow under its influence. When there are no mitral symptoms it seems to be on the whole unwise to use *digitalis* and thereby increase the length of diastole, but the evidence on this point is conflicting. I have used *sodium formate* (15) in doses from 5 to 10 grains for children with, I think, some benefit, from its tonic action on the cardiac muscle.

2. The arterial type. The arterial cases are more unsatisfactory.

High blood-pressure must be regulated by the addition of *iodide of sodium* to a cardiac tonic such as *tincture of nux vomica*; or *nitro-glycerine* should be added to *strychnine* in 1- to 3-minim doses of the solution. Pain if paroxysmal must be dealt with, as in the treatment of angina pectoris, Chapter xiv, p. 247. If more persistent, *morphia* cautiously given is of great value.

Special directions should be given to these patients as to the avoidance of *sudden imprudent exertion*, such as running for a train after meals, jumping out of bed in the morning, hurrying up stairs, and the great importance of a quiet life must be insisted upon.

D. Aortic Stenosis

There are no special points in the treatment of this affection ; whenever there is a sign of failure, the force of the heart must be strengthened by *rest* and *tonics*, and the peripheral resistance lowered by *eliminants* and *vaso-dilators*. Pain must be combated as in aortic regurgitation.

The tricuspid and pulmonary lesions require no special mention, for the indications are included under the mitral lesions.

CHAPTER X

ACUTE AND SUBACUTE PERICARDITIS (NON-RHEUMATIC)

Aetiology—Suppurative pericarditis—Morbidity—Pyopneumo-pericardium—Symptoms—Diagnosis of pericardial effusion from cardiac dilatation—Prognosis—Treatment—Tuberculosis of the heart—Pericarditis—Endocarditis—Serositis.

Among the causes are :—

1. Many infective diseases other than rheumatism. As examples may be given the 'septic', including the pneumococcal, and gonorrhoeal, also the tubercular and syphilitic infections, and possibly the scarlatinal.

2. Renal diseases.

3. Direct extension of infection from a neglected empyema, or from an hepatic or subphrenic abscess.

4. Rupture of an intra-pericardial aneurysm, producing sometimes inflammation, but more usually haemo-pericardium.

5. Traumatic injuries.

6. Gout and diabetes, leukaemia, lymphadenoma, and other disordered blood states, are sometimes complicated by pericarditis.

7. New growths.

I (a). SUPPURATIVE PERICARDITIS

This name is sanctioned by usage for those forms of pericarditis in which the exudation generally becomes purulent. The name is not a good one, for it lays too great a stress upon

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suppuration, which is no more than a peculiar phase of inflammatory reaction. There are many kinds of pus formation; and rheumatic pericarditis may, as has already been pointed out, reach this stage, and on the other hand those very infections which have been termed suppurative, may cause a virulent and rapidly fatal pericarditis, and yet the fluid in the pericardium be clear.

Suppurative pericarditis is less common than the rheumatic form, and generally occurs in association with *pulmonary disease*. Infections with the pneumococcus, the staphylococci, or the varieties of pyogenic streptococci, are among the most frequent exciting causes. Such infective diseases as measles, whooping cough, and influenza, predispose to its occurrence, as do also marasmic conditions. This form is most frequent in *childhood*, and 80 per cent. of the cases under twelve years of age are associated with pulmonary disease. In 60 per cent. the pulmonary affection is an empyema. Local abscesses, osteo-myelitis, and general pyaemia are among the rarer causes.

In contrast to the rheumatic form, *valvular disease* is not so frequently associated, and there can be no doubt that the pericarditis itself is much more frequently than in rheumatism a secondary lesion, although sometimes the pneumococcus may attack many serous membranes simultaneously, and among these the pericardium.

Morbid anatomy. The condition of the pericardium differs considerably. In very acute cases, and in those associated with extreme marasmus, there may be a *clear fluid* with only a few flakes of exudation, in other cases the condition may very closely resemble a *sero-fibrinous exudation*. Again, the pericardium may be greatly thickened, or there may be a large collection of *pus* distending the sac and compressing the heart. In some exceptional cases the pus has

perforated the pericardium. The fluid is usually sweet, but sometimes foetid. Cases are on record in which the fluid has been absorbed, leaving behind a pultaceous mass. The heart itself may not be dilated, and there is not the frequency of dilatation that occurs in rheumatism.

Pyo-pneumo-pericardium. This will be the most convenient place in which to mention this rare occurrence. Injury from stabs, or fractured ribs, may be causes, or it may result from disease placing the pericardial sac in communication with a tube or cavity containing air. Cancer of the stomach or oesophagus, eroding the pericardium, or a pulmonary cavity rupturing into it, are examples of this latter occurrence. There is some uncertainty as to whether a pericardial effusion itself can by decomposition produce gas within the sac. The fluid is generally, but not invariably, purulent; should it be clear the condition is termed hydro-pneumo-pericardium.

Symptomatology of suppurative pericarditis. The *symptoms* are often exceedingly vague, and in childhood it is *most difficult* to lay the correct stress upon such as are present, for two reasons. One is the usual absence of pericardial friction, the other the almost invariably co-existing pulmonary disease which draws the attention away from the heart.

Wasting, pallor, irregular panting respiration, orthopnoea, vomiting, and fainting attacks have often been recorded. The temperature may be raised or subnormal.

Physical signs. Great rapidity of the *pulse* with irregularity, progressive increase in the cardiac dulness, with possibly some bulging of the precordial region, and more significant still, gradual *muffling* of the heart sounds are among the most important physical signs.

In suppurative pericarditis very large effusions have been

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recorded, and this leads to the consideration in detail of the evidences of a large pericardial effusion.

Clinical Evidences of Extensive Pericardial Effusion.

1. *The pulse* is rapid, and the wave small, ill-sustained, and sometimes very irregular.

2. There may be some bulging of the precordial area, and the intercostal spaces, especially on the left side, may be widened. A prominence may also appear in the epigastrium.

3. *The cardiac impulse* is either absent, or there may be an undulatory movement visible over the third and fourth left intercostal spaces. This impulse is not caused by the apex, but by a portion of the heart wall nearer its base.

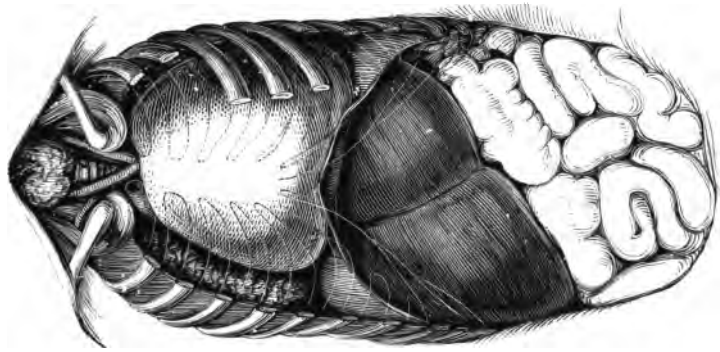
4. The area of precordial dulness is strikingly increased. It will be remembered that there are certain places at which the pericardium fits loosely and where fluid can collect. The right and left lower angles are two of these, and a third is between the large vessels and the pulmonary conus. Further, the inflammation of the parietal pericardium weakens that structure and causes it to yield to any undue pressure. Lastly, at the back of the heart there may be a considerable collection of fluid. The results of a considerable effusion are : (1) An extension of the dulness upward and laterally. The extension upwards may be so great as to produce absolute dulness up to the left clavicle, the upper lobe of the left lung being pushed aside and compressed. (2) An alteration in the shape of the precordial dulness. The outline of a large effusion is *pear-shaped*, and can be also compared to an ice-bag filled with fluid ; the metal top representing the large vessels. Great importance has been rightly attached to this physical sign ; but it is not pathognomonic. Extreme dilatation of the heart with a small quantity of effusion will

PLATE X



A

Suppurative pericarditis, the result of a pneumococcal infection. The pericardium has been reflected.
From Curr's 'Practice of Paediatrics'; by permission of Messrs. Lea Brothers.



B
 Extensive pericardial effusion.
By permission of Messrs. Macmillan. From Sibson's Collected Works.

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produce a similar outline, as I have proved at the bedside and checked later in the post-mortem room. For this reason I am unable to accept as of very great value the presence of dulness on percussion in the fifth intercostal space on the right side.

5. The *absolute dulness* that is present over a large effusion, and the abrupt transition to pulmonary resonance, are suggestive. The rapidity of the enlargement, on the other hand, may be equalled in acute dilatation.

6. The most valuable auscultatory signs are the *progressive muffling* and sometimes *entire loss* of the cardiac sounds. The impression that is given the ear is a very different one from the feeble yet clear heart sounds of extreme dilatation.

7. Ewart, who has investigated the clinical signs of pericardial effusion with great care, attaches much importance to a modification in the respiratory signs over the base of the left lung posteriorly. He finds there a *square patch of percussion dulness* bounded above by a horizontal line drawn outward from the vertebral column at the level of the ninth or tenth ribs, and ending just within a vertical line dropped from the vertebral angle of the scapula. The upper and outer boundaries are abrupt, and below this the dull area reaches to the base of the chest. It usually also extends, though less definitely, for a short distance to the right of the vertebral column.

Over the dull area on the left side the breath sounds are absent.

8. *Oedema of the skin* over the precordial area may be present when there is pus in the pericardial cavity.

Pyo-pneumo-pericardium. When there are both air and fluid in the pericardial sac the physical signs differ in some respects, and these differences will lead to the diagnosis.

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Thus :

(1) On palpation a *crackling sensation* may be felt synchronous with the movements of the heart.

(2) The note on percussion will be *tympanitic* and not dull.

(3) *Changes of position* will alter the relation of the gas and fluid to one another.

(4) The heart sounds may have a *ringing character*, and various metallic and splashing sounds are audible, the results of the churning together of gas and fluid in the pericardial sac.

From the foregoing account of the physical signs of suppurative pericarditis, it is evident that if there is the *usual absence of pericardial friction* and a *scanty effusion*, the detection of the disease is almost impossible.

The course of all these cases is as a rule fatal, although occasionally surgery has saved a life, and there are some few that have apparently recovered by the '*vis medicatrix naturæ*'.

Diagnosis. If there is a large pericardial effusion this must be first of all distinguished from *extreme cardiac dilatation*.

The important points of distinction are :—

1. The history of the illness. The illness which produces dilatation is often a more gradual one, although both rheumatism and influenza are notable exceptions.

2. The development of the symptoms. This may be a most valuable assistance. A progressive muffling of the cardiac sounds synchronous with enlargement of the cardiac area, and marked percussion dulness over the pericardium, greatly favour pericarditis with effusion.

Clear, weak cardiac sounds with a puffing systolic murmur are in favour of dilatation, and this likelihood is the more

probable if progressive shortening and enfeeblement, rather than muffling of the sounds, have been noticed.

3. A rapid and extensive increase of the cardiac dulness upward toward the left clavicle favours the diagnosis of effusion.

4. In moderate dilatation, the inward curve of the right auricle as it passes downward and leftward to join the right ventricle can generally be detected in children by percussion. In pericardial effusion the distended sac obliterates this curve, and substitutes for it a line extending from above downward and progressively outward, the result of distension of the right lower angle of the pericardium.

This sign, described by Ewart, fails in great dilatation, for then the right auricle is so greatly distended that its right border has the same inclination as a distended pericardium, namely, downward and outward.

5. The transition from cardiac dulness to pulmonary resonance is more abrupt in effusion than in dilatation, and the sense of resistance to the finger greater over a large effusion than it is over a dilated heart.

A *localized pleural effusion* on the left side may closely simulate a pericardial effusion by pushing the heart somewhat to the right, and overlapping it in front. The diagnosis rests chiefly upon the nature of the illness. If there is apparently a large pericardial effusion, and yet the cardiac symptoms are conspicuous by their absence, this should suggest the possibility of an extra- rather than an intra-pericardial effusion. Exploration may, however, be the only solution.

Where there is neither friction nor considerable effusion suppurative pericarditis may be either overlooked or is liable to be mistaken for *tuberculosis* of the lungs.

Attention must be particularly directed to great rapidity

174 ACUTE AND SUBACUTE PERICARDITIS

and irregularity of the heart, to vomiting and livid pallor, panting respiration, orthopnoea and syncopal attacks.

The prognosis is exceedingly grave, and at the present time the hope lies in surgery combined with early diagnosis.

Treatment. Two surgical procedures can be adopted, paracentesis of the pericardium, and incision and drainage.

The first measure is indicated when from the urgent condition of the patient and embarrassment of the heart the major operation is contra-indicated. By means of it the pressure can be relieved, and an opportunity is thus given for the circulation to recover. With improvement the complete operation is undertaken.

In operations upon the pericardium it is necessary to remember that it is in only a percentage of cases that the left pleura leaves the middle line at the level of the fourth costal cartilage, and that it is frequently not reflected until the level of the fifth or sixth costal cartilage.

Paracentesis may be undertaken by the introduction of the needle in the left costo-xyphoid angle ; it should graze the lower end of the body of, the sternum, and pass up and inward behind it to the cavity of the pericardium.

Another position often chosen is the fifth left interspace half an inch to the left of the sternal border.

The general management of the patient in other respects resembles that described under the palliative treatment of acute rheumatic pericarditis.

I (b). TUBERCULAR PERICARDITIS AND TUBERCULOSIS OF THE HEART

This will be a convenient occasion to deal with this subject. Cardiac tuberculosis as a clinical disease is not common. Sometimes in the *post-mortem* room, tubercles are found in both the endocardium and pericardium as

PLATE XI



**Tubercular endocarditis, from the Museum
of Great Ormond Street.**

By permission of Messrs. Lea Brothers.

a part of a general miliary tuberculosis, but by far the most important lesions are:—

1. Acute, subacute, and chronic pericarditis.
2. Chronic endocarditis.
3. Multiple serositis.

1. In **acute tubercular pericarditis** there may be very extensive effusion of a clear or blood-stained fluid with all the attendant consequences, and in such cases it may be necessary to tap the pericardium. There is nothing characteristic in the symptoms, but should there be no history of rheumatism or 'septic' infection, and other evidences of tuberculosis be detected, the diagnosis may be plain. In some of these cases, as happens with the pleura, the fluid reaccumulates, and the illness drifts from the acute into the subacute stage.

In other cases the disease is never acute. There may be slight fever and precordial pain with the appearance of pericardial friction, and the condition is probably thought to be a subacute rheumatic attack. Later the case appears as one of *adherent pericardium* or *multiple serositis*.

These cases give us a warning not to accept all cases of pericardial friction as necessarily rheumatic.

2. **Tubercular endocarditis** is usually a chronic insidious process, the mitral, aortic, and tricuspid valves may all be affected, and there may be extensive calcification both of the valves and the valve rings.

When *malignant endocarditis* arises, although there is no reason why it should not be tubercular, it would appear to be more often the result of a mixed or added infection.

Neither the chronic nor malignant types show any characteristic features, but there may be overwhelming evidence of tubercle elsewhere and none of any other infection, and guided by this, a correct diagnosis may be made.

3. Multiple serositis. This may undoubtedly result from tuberculosis, and some writers believe it to be the most frequent cause. I have seen four undoubted cases, each one resembling the other very closely.

It is difficult, as it is with rheumatism, to explain why the condition does not arise more frequently, but the difficulty is not so great because the pericardium is far less frequently affected in tuberculosis than in rheumatism.

The onset is insidious. It may follow acute pericarditis after a considerable interval, or no history of an illness may be obtained until the patient appears with ascites.

Symptoms. The symptoms are *shortness of breath* and *cyanosis, wasting*, and sometimes *multiple effusions*.

Children and young persons are most often attacked. There are frequent complaints of pain in the chest and abdomen on account of recurrent focal pericarditis, pleurisy, or perihepatitis.

There may be signs of an adherent pericardium, and of thickened pleura or pleural effusions, as in the rheumatic form. The liver is large and congested, and sometimes enveloped in a case of inflammatory exudation, a condition sometimes termed 'the iced liver'.

The course is a long one, and eventually there is great wasting. There is one feature about these cases which distinguishes them, when it occurs, from the rheumatic cases, and this is the sudden supervention of *cerebral symptoms*. In three fatal cases in my experience this has occurred, and the explanation was in each case the same, viz. the supervention of *tubercular meningitis*.

The diagnosis is not always easy.

The chief conditions to differentiate are:—

- i. Tubercular peritonitis with ascites.
- ii. Cirrhosis of the liver with ascites.

- iii. Adherent pericardium only, with ascites.
- iv. Chronic peritonitis of unexplained origin.
- v. Congenital visceral syphilis.
- vi. Renal disease with ascites:
- vii. Abdominal lymphadenoma.

If there are evident signs of an adherent pericardium, uncomplicated tubercular peritonitis, and hepatic cirrhosis can be excluded. The urine must be examined, and search made for signs of congenital syphilis and lymphadenomatous glands.

If there is distinct evidence of pleurisy or slight irregular fever, with focal exacerbations of pleurisy or pericarditis, simple adherent pericardium can be dismissed.

The prognosis is bad, and the treatment palliative.

Affections of the pericardium in renal disease are considered under the renal cardiac affections, Chapter xiii.

Pericarditis by *direct extension* presents no very special features, but before the inflammation becomes actually intra-pericardial, pleuro-pericarditis may occur, and its recognition may save the greater disaster. Before the operative procedure for empyema was thoroughly established this form of pericarditis was much more frequent than it is now.

Intra-pericardial leakage of an *aneurysm* may produce pericarditis, but the rupture generally produces rapid death, with precordial pain and sudden collapse.

Pericarditis from *traumatism* in *gout*, *leukaemia*, and other *blood diseases*, is in no way characteristic, and although pericarditis in connexion with new growths may be remarkably latent, this again needs no further consideration here.

CHAPTER XI

ACUTE ENDOCARDITIS (NON-RHEUMATIC)

Simple endocarditis—In scarlet fever—Acute rheumatoid arthritis—Chorea—Malignant endocarditis—Causation—Morbidity—Anatomy—Symptoms—Diagnosis—Prognosis—Treatment.

THIS may be simple or malignant:—

A. SIMPLE

Simple endocarditis may follow many infections other than the rheumatic, but is decidedly rare. One great exception is scarlet fever, but this form, if not actually rheumatic, is very closely allied. Endocarditis, when associated with chorea, I look upon as rheumatic. Among other infections may be mentioned syphilis, diphtheria, influenza, and measles. Endocarditis has also been recorded in gout, diabetes, and in some cases of renal disease. Another fact of interest is the occurrence of simple endocarditis in acute rheumatoid arthritis.

Heart Disease in Scarlet Fever. There are two types: one due to a streptococcal or micrococcal infection of the heart; the other associated with acute renal disease, vide Chapter xiii, p. 214.

These cases are clinically indistinguishable from those produced by the rheumatic infection, and in one such case I investigated with Paine we were unable to differentiate

the streptococcus from the micrococcus of rheumatism by the ordinary morphological and cultural tests or by the experimental results. When to this is added the fact that multiple arthritis, chorea, and nodules may all follow scarlet fever, it is certain that this post-scarlatinal condition, if not identical with rheumatism, is exceedingly closely allied.

Pericarditis, *endocarditis*, or *myocarditis* may follow scarlet fever, or, as in rheumatism, there may be a general *carditis*.

It seems probable that the infection in scarlet fever dates from the primary or secondary sore throat, and the rapidity of the appearance of the symptoms will depend upon the severity of this infection. When the scarlet fever is virulent, *fatal pericarditis* is not very unusual, and associated with it there may be pleurisy and nephritis. Such cases may arise in the second week after the appearance of the rash, or as late as six weeks, and may prove rapidly fatal in a week or fortnight; the symptoms resemble those of malignant rheumatic pericarditis.

The *symptoms* of the less severe forms of *carditis* are practically the same as those of rheumatism, and show the same tendency to relapse.

Mitral regurgitation and stenosis, aortic regurgitation and combined mitral and aortic lesions may all occur, and in some cases, as in rheumatism, the endocarditis is malignant.

Chorea is very frequently associated with *carditis*, and particularly in females with mitral stenosis; this heart disease I believe to be rheumatic, and its clinical character accords in every respect with this view.

Acute Rheumatoid Arthritis. In this affection—of doubtful definition—*endocarditis* may certainly occur resembling in distribution and character that of acute rheumatism.

In addition, in chronic arthritis deformans, or as some prefer, chronic rheumatoid arthritis, a frequent occurrence

is a rapidly acting, feeble heart *without any valvular lesion*. This is generally looked upon as a neuro-muscular affection. The symptoms are, cold extremities, some breathlessness on exertion, and palpitation; examination of the heart shows a feeble impulse with irritable action and short clear sounds. Simple endocarditis following other infections shows no characteristic features.

B. MALIGNANT ENDOCARDITIS

Among these non-rheumatic cases are found some of the most virulent types of this disease.

When a broad view of the problem of infective endocarditis is taken, the general impression received is that when a micro-organism such as the pneumococcus, which generally spares the heart, does attack the valves, the tendency is to produce a malignant condition. The same is true of the septic streptococci. When these cause a fatal septicaemia there is great enfeeblement of the myocardium, but when they form a local focus in a valve the tendency is to malignancy.

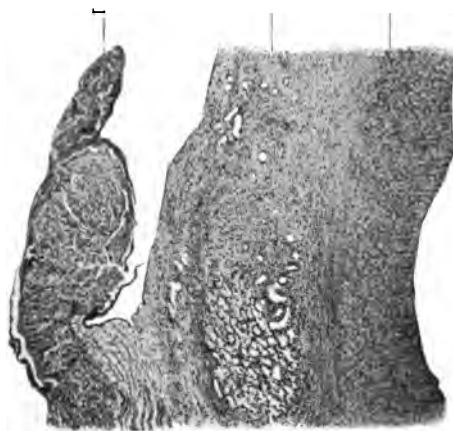
It is important to recognize that a great number of different bacteria may on occasion attack the valves and produce malignant endocarditis, and also to realize that the lesions produced by these various micro-organisms are very similar in character.

Reference must be made to larger treatises for details, here for the purposes of illustration may be mentioned certain groups of infections.

(1) A comparatively well-defined group containing: the pneumococcus and streptococci of the pyogenic type; the staphylococci, meningococcus, influenza bacillus, gonococcus, typhoid bacillus, and bacillus coli.

(2) A less well-defined group containing: the bacillus

PLATE XII



A To illustrate the essential difference between a vegetation in

Simple endocarditis.



B

Malignant endocarditis.

1. The necrotic area of the vegetations. In figure B there is a dark fringe in this area, which is produced by masses of streptococci; there are no visible micrococci in the corresponding area of figure A.

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To face p. 180]

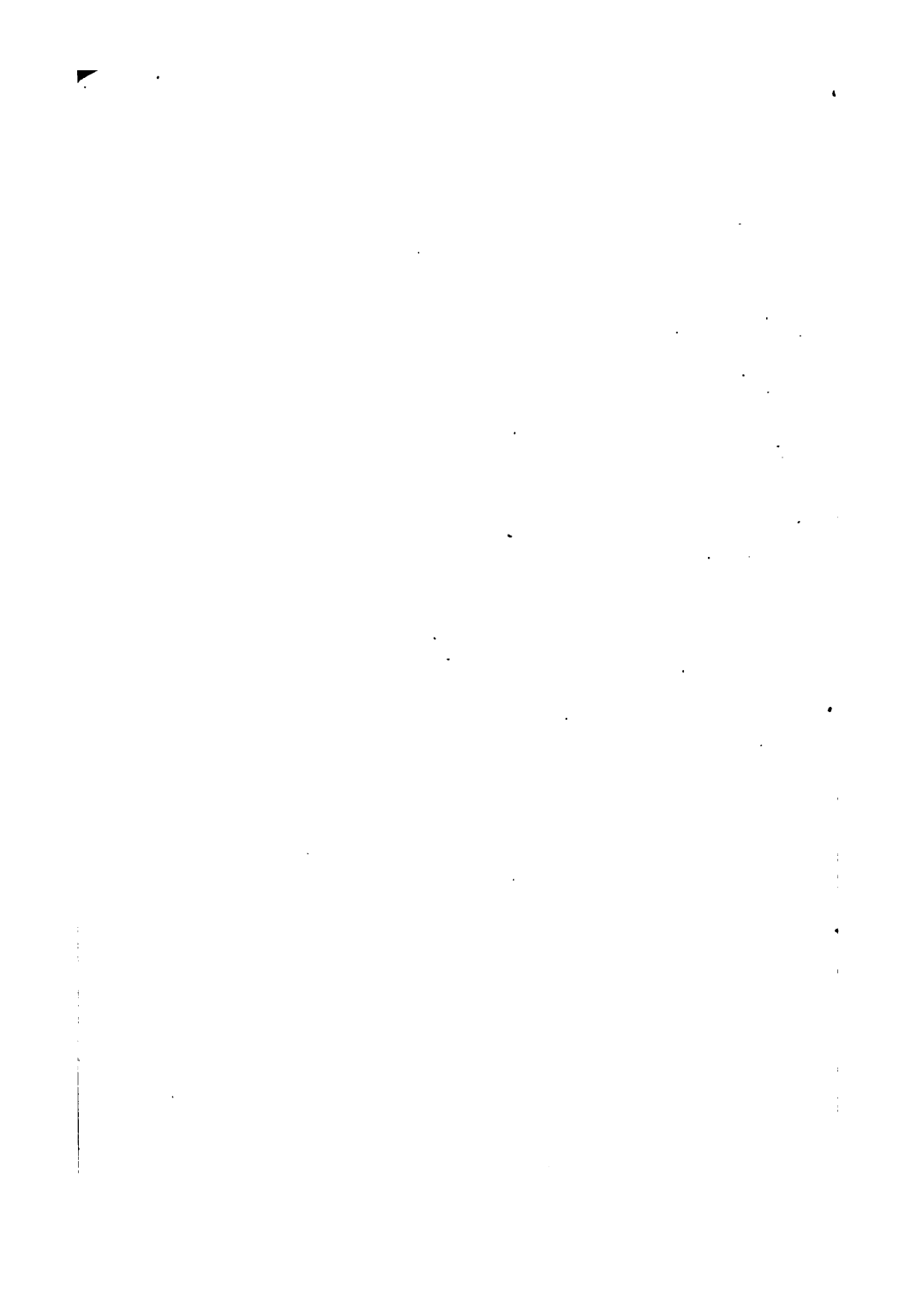




PLATE XIII



A

A white infarct in the kidney of a rabbit, produced by intravenous inoculation of the diplococcus rheumatis. There was active mitral endocarditis.



B

Malignant endocarditis. A part of the left ventricle and the commencement of the aorta are open. There is a large fungating vegetation on one of the aortic cusps.

From the Museum of University College Hospital.

endocarditis griseus, micrococcus endocarditis, bacillus endocarditis capsulatus, &c.

(3) The diplococcus rheumaticus, which has been already considered.

(4) Mixed infections.

Certain *predisposing factors* can be traced in this disease. Young adult life is the most favourable time for its occurrence. Anaemia, bad sanitation, dark, damp dwellings or workshops, debility from influenza and previous rheumatism, and the puerperal state favour the occurrence.

The Morbid Anatomy. The *most virulent forms* are those in which vast numbers of the infective agent are found in vegetations which may be small in size. In other cases the vegetations are *exuberant* and spread on to the auricular and ventricular endocardium. In yet other cases the *ulcerative processes* predominate, and the valves are perforated or severed from their chordae tendineae. Mural vegetations may perforate the wall of the heart or cause an aneurysm. *Masses of micrococci* may be found in the peri-vascular spaces in the *cardiac* walls, and intense myocarditis result from their presence. The pericardium may be infected and *pericarditis* result. Lastly, fragments of vegetations are cast hither and thither by the blood-stream, causing *embolism* in various organs and structures of the body.

The *vegetations* consist of necrotic tissue containing bacteria, and upon them are often deposited layers of fibrin from the blood. The base of the vegetations is infiltrated by leucocytes, and gradually merges into the tissue of the valve. In some of the most virulent types of infections these vegetations are very soft and friable.

In some cases also the emboli produce *local abscesses* and sometimes extensive suppurations, but compared with the frequency of infarction these suppurative processes are

unusual. Severe damage to the valves on the *right side of the heart* occurs more frequently in this malignant type than in the simple one.

It is a remarkable fact that even in these cases of severe infection it is often difficult to isolate the infective agent from the blood, and it may be equally difficult to ascertain the site of entrance of the infection into the blood.

There are undoubtedly a great many *paths of invasion* : among these the throat, the bones as in septic osteo-myelitis, the uterus as in some puerperal infections, the lungs or pleurae, the gall bladder, the intestines, and the urethra may be mentioned.

Malignant endocarditis is in these cases a secondary development, but in some cases it may be, so far as can be detected, the *primary seat* of infection.

All writers have called attention to the frequency with which in this disease the valves have been the *seat of previous inflammation*. This occurrence suggests that, rather than a preference on the part of the micro-organisms for indurated scar tissue, there is either a lack of resistance in this tissue, or the micrococci have been lying latent in some half-healed patch and have become once more and this time unusually virulent.

Symptomatology. This varies with the virulence and nature of the infection. In some cases the general poisoning is severe, and the *temperature* is high and sustained. There are rapid *emaciation* and profuse *sweating*, with progressive *anaemia*. The *tongue* is dry and coated, and the abdomen distended. In such cases also there may be severe *diarrhoea*. The *respiration* is rapid and panting. There are *asthenic delirium*, and sometimes *purpura*, sanious bullae and erythematous eruptions, with *albumin* and *blood* in the *urine*.

The physical signs. It is most remarkable how *latent* the cardiac physical signs may be. The heart is *excited* and the *pulse rapid* and feeble, but no dilatation or murmur may be detected. If, however, death is not very rapid, a *systolic or diastolic murmur* usually appears in the mitral tricuspid or aortic region, and the heart commences to dilate. The *spleen* generally enlarges, and multiple emboli may occur. *Death* sometimes occurs in under a week, or more usually life is prolonged for some weeks, but the course of the

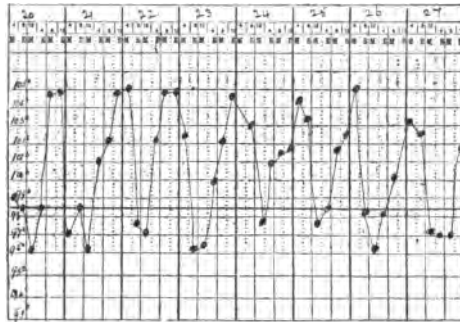


FIG. 19. Malignant endocarditis of the pyaemic type.
Chart illustrating the irregular fever.

illness is progressively down-hill. This virulent type is of rare occurrence, the only primary one of this nature in my experience was the case of an infant of eighteen months who succumbed to a malignant endocarditis of mysterious origin.

In other cases *rigors* and irregular *intermittent pyrexia* are prominent features. The temperature may range through seven or more degrees in the day, and for weeks there may be a continuance of the oscillations with the greatest regularity.

Emboli may be numerous, *retinal haemorrhages* may occur, and the *spleen* reach a great size. *Meningeal* and other severe *cerebral* symptoms sometimes develop, and malignant endocarditis is the commonest cause of *cerebral haemorrhage* in childhood.

In such cases the cardiac signs are generally more obvious, and two valuable diagnostic indications may be detected.

The first, a marked change in the character of the murmur at one of the valvular orifices ; *the other*, the development of a progressive lesion at the site of another valve while the patient is under observation.

Lastly, the disease may run a very quiet subacute course, and no suspicion be aroused until an embolus and some irregular fever warn us that the heart disease is advancing and is active in character. Should the vegetation be on the endocardium rather than the valves the physical signs are mostly negative.

Diagnosis. The history and course of this disease have been altered by the demonstration and recognition of the transition of simple into acute endocarditis, for it is now clear that there is every grade between the two types.

The diagnosis consists of *three steps* :—

The first is the decision that the condition is one of acute valvular disease.

The second, that it is malignant in type.

The third decides the nature of the infection.

The *typhoid form* is the most difficult, and may be easily mistaken for acute septicaemia, acute general pneumococcal infection, cerebro-spinal fever and typhoid fever, or acute lymphadenoma. The *septic form* may be mistaken for general pyaemia or malaria. The great points are to determine that the heart is actively damaged, and further,

locally damaged in the position of the valves. Next, search should be made for evidence of embolism, and lastly, the other diseases must be taken one by one and considered in detail.

The prognosis is most serious, but how serious is hardly known, because the rigid mental line between simple and malignant endocarditis has not yet been broken down. Cases which follow rheumatism (not necessarily rheumatic themselves) are the most likely to recover, and particularly if they are subacute and without profound constitutional poisoning. The recovery may be extremely slow, and the heart much damaged. Primary and secondary cases in no way associated with rheumatism are usually fatal.

Treatment. Open-air treatment is advisable if the weather and surroundings are suitable for this purpose. Food should be plain, ample, and generous so long as the patient has the capacity for assimilation.

Special treatment. After a review of the reports upon the treatment of malignant endocarditis by *antistreptococcus serum*, imperfect though many are, I think if a streptococcus is isolated from the blood, a serum should be tried. The *polyvalent sera* appear to be the best. This one would expect at the present time, when the problem of the streptococci is so complex and little understood. If it is decided to use this method of treatment, there should be no delay, as so often occurs when a measure of uncertain value is employed. We have at any rate the right to tell our patients that remarkable recoveries have occurred when this serum has been used.

Method of administration. Dyce Duckworth and others have recorded good results with rectal injections of serum. Sera thus administered certainly get into the system, for eruptions follow their use by the bowel.

When the patient is much distressed by subcutaneous injection, this method is indicated, but the subcutaneous method appeals to me as a more thorough one, and Newton Pitt has recorded some striking recoveries in the Transactions of the Royal Medico-Chirurgical Society, vol. 88.

Subcutaneously the dose of the polyvalent serum is usually 10 c.c., repeated every day for three or four days and then at longer intervals. Rectally, double this dose is indicated. *Quinine, strychnine, arsenic, and iron* may all be of some service in improving the patient's appetite and anaemia, but too often they are utterly useless for arresting the disease. *Nuclein* preparations have been used, but the results have been disappointing.

Recently a *vaccine treatment* after the method of Wright has been credited with success. The particular infection must be isolated and the vaccine prepared from it. The effects of this are checked by observations upon the opsonic index for the particular micro-organism.

The method is too recent in its employment for any judgement to be formed.

CHAPTER XII

MYOCARDIAL AFFECTIONS (NON-RHEUMATIC)

Causation—Acute myocardial affections—Diphtherial heart disease—Morbidity—Anatomy—Symptoms—Diagnosis—Prognosis—Treatment—Influenzal heart disease—Morbidity—Anatomy—Symptoms—Diagnosis—Prognosis—Treatment—Septic myocarditis—Symptoms and treatment—Summary of pathological changes—Chronic myocardial affections—Causation—Morbidity—Anatomy—Diseases of the coronary arteries—Symptoms and results—Fatty heart—Other causes of—Morbidity—Anatomy—Symptoms—Diagnosis—Prognosis—Fibroid heart—Symptoms—Diagnosis—Prognosis—Fatty infiltration of heart—Causation—Symptoms—Prognosis—Syphilitic disease of the heart—Symptoms—Diagnosis—Prognosis—Treatment—Heart in anaemia—Symptoms—Diagnosis—Prognosis—Strain of the heart—Severe form—Mild form—The heart in alcoholism.

IN this group there are included some very important forms of heart disease, which clinically fall into two great groups, the acute and chronic.

The causes of myocardial disease may be classified in the following way :—

1. (a) Those due to the action of bacterial poisons, as for example the rheumatic, scarlatinal, diphtherial, influenzal, syphilitic, pyaemic, and others, including among tropical diseases beri-beri.

- (b) Those due to chemical poisons, e.g. phosphorus and alcohol.

2. Those due to impaired nutrition of the muscle, the result of coronary disease.

3. Those due to altered conditions of the blood, as in Addison's disease, anaemia, and leukaemia.

4. Those due to over-exertion, mental, physical, and emotional.

5. Those due to increased peripheral resistance:—

(a) In the systemic circulation.

(b) In the pulmonary circulation.

6. Those due to growths.

7. Those due to parasites.

A. ACUTE MYOCARDIAL AFFECTIONS

The two forms most commonly met with are the diphtherial and influenzal.

I. DIPHThERIAL HEART DISEASE

Endocarditis and *pericarditis* are rare, and their occurrence raises the suspicion that there is a multiple infection.

The usual type is a *myocardial affection*. This is not, as a rule, a diphtherial paralysis, but the result of the action of the extra-cellular toxins of the diphtheria bacillus upon the cardiac muscle.

Morbid Anatomy. There is often very little, if anything, to be seen on *macroscopic examination*.

The cardiac muscle may appear pale and flabby, and break down easily, but otherwise no change may be observed.

Microscopic examination, on the other hand, may show profound changes. Some fibres are completely destroyed, in others the nuclei are swollen or shrunken, and show hyperchromatosis; the striation of the fibres is more or less completely lost, and the shape of the fibres is irregular. The most definite of all changes are *fatty*; the deposit may be in fine specks or in larger irregular droplets. The

interstitial tissues show little; some increase of cellular elements and minute haemorrhages in the region of the small blood-vessels are the most frequent changes.

Among the recent workers upon this subject, C. Bolton and L. Dudgeon have independently made valuable contributions. Bolton has shown that in some cases there are

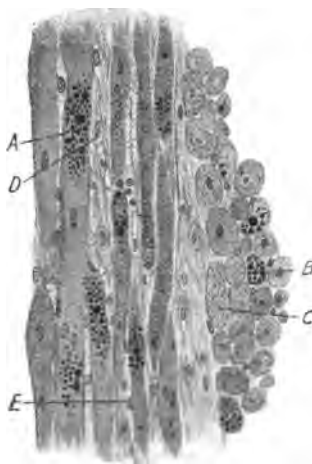


FIG. 20. A section through a part of the left ventricle of a case of diphtherial heart disease, fatal on the seventeenth day. A. Muscle fibre showing much localized fatty change. B. Another fibre in transverse section. C. Transverse section of muscle showing complete destruction. D. Longitudinal section showing complete destruction. E. Cellular elements between the muscle fibres. (Reproduced by permission of the *Lancet*.)

acute degenerative changes in the medulla oblongata affecting the sensory and motor nuclei of the vagus, but he found no changes in the vagus nerves.

Dudgeon has amplified our knowledge of the myocardial changes by the use of special staining reactions, and has

brought forward some evidence to show that antitoxin has a preventive effect upon their occurrence.

Symptomatology. For clinical purposes two forms will be considered :—

1. The Severe. 2. The Mild.

1. The severe form is fortunately rare. This group contains those in which the effects of the diphtherial toxaemia are widespread, and damage, in addition to the heart, the respiratory apparatus. It may happen that the nature of the sore throat has been overlooked, and no fears are aroused until the child (for children are the most frequently affected) is noticed to *squint*, or to be unable to read. *Liquid food regurgitates*, and there may be *ataxy* and *fainting fits*. A symptom of grave importance is an *ineffectual cough*, which points to a commencing paralysis of the diaphragm. Examination will show that the diaphragm does not move with respiration or that its action is reversed.

The physical signs. The *pulse* is rapid, feeble, and irregular.

The impulse of the *heart* is weak, and there is some dilatation. The *sounds* are short, sometimes approximated, and a soft systolic mitral murmur may make its appearance.

The urine generally contains albumin.

Death may be rapid from sudden arrest of the heart, or from a combination of respiratory and cardiac failure.

2. The mild form. This constitutes by far the larger group. The first evidences usually show themselves within the *first four weeks*, but they may occur in the first few days. Dudgeon's observation, that experimental fatty changes may be noticed in sixteen hours, is of great interest in view of this, and particularly as such fatty changes have been actually demonstrated in fatal cases in man as early as the third day.

Symptomatology. If the child is under treatment in bed the symptoms are but slight. *Pallor* and *muscular lassitude* with *loss of appetite* are the most frequent. If the patient has not been confined to bed *syncopal attacks* may occur.

The physical signs. The *pulse* and *heart* need careful examination. The pulse may be rapid, or slow, or again irregular.

The *impulse* is feeble and displaced outwards, the area of deep cardiac dulness is slightly increased. The first sound at the apex is short, and the second at the pulmonary base is accentuated.

A *soft systolic murmur* is sometimes audible internal to the nipple.

Diagnosis. The condition is at first very unobtrusive, and herein lies the danger. The disappearance of the *knee jerks* is sometimes helpful when suspicion is aroused. Any cause of cardiac dilatation will have to be considered, but those most likely to need differentiation are influenza, rheumatism, and renal disease.

The other evidences of diphtheritic paralysis must be sought, the history investigated, and the Klebs-Loeffler bacillus if possible cultivated from the throat.

Immediate prognosis. In the severe cases this is always very grave, and if the heart is damaged in the first ten days the outlook is more serious than in the usual mild forms. These latter generally recover if recognized early and correctly treated.

Dangerous symptoms are the occurrence of *respiratory paralysis*, *severe vomiting*, *great pallor*, *restlessness*, and *syncopal attacks*.

Physical examination is of some assistance. A rapid and feeble or a very slow pulse is serious, and *approximation*

of the cardiac sounds is particularly ill-omened. The worst cases may show very little dilatation.

Remote prognosis. This is good, and the rule is complete recovery. There are, nevertheless, cases which behave exactly like some of the rheumatic, and remain breathless, and seriously damaged for months and even years. On any exertion there is palpitation, and the general health suffers in consequence. In some adult cases there may be attacks of angina, and great functional disturbance.

Treatment. *Good nursing* is a great assistance in the management of cases in childhood. Absolute *rest* is the most important point, and the experience at the Children's Hospital, Great Ormond Street, has been that when the children are secured early in the attack of diphtheria, and treated by careful rest and suitable doses of antitoxin, the alarming cases of heart failure do not occur.

The *diet* must be light and fluid, and, if there is difficulty in swallowing, nasal feeding may be necessary. These feeds must not be bulky, nor given too hurriedly. The bowels must be kept gently open, and in severe cases enemata are preferable to purgatives.

The drugs most relied upon are *atropine* (16) and *strychnia*, and later *iron* and *nux vomica* (17), or *quinine*.

Such drugs as *digitalis* and *strophanthus* are to be avoided.

However mild a case of diphtheria may be, *three weeks* absolute rest is advisable, on account of the cardiac complications, and each forward step should be gradual, and gauged by its effect upon the heart and pulse.

For severe cases prolonged rest may be needed, and if chronic irritability and weakness of the heart remain, *passive movements* and *massage* and *graduated exercises* should be employed, until the time for ordinary voluntary movement is reached.

II. INFLUENZAL AFFECTIONS OF THE HEART

Influenza may attack the heart at any age, but it is particularly liable to produce disastrous results in the elderly or in those who have already a crippled heart.

Morbid anatomy. Such pathological changes as have been chiefly observed are of the nature of *parenchymatous fatty degeneration of the myocardium* with resulting dilatation. Purulent pericarditis has been recorded, but it seems probable that this is in most instances the result of a secondary infection from a complicating pneumonia or empyema. *Endocarditis, both simple and malignant*, are also occasional events. Clinical observation, however, makes it apparent that in many instances the chief injury has been to the *nervous mechanism* of the heart.

Symptomatology. In *infancy* influenza may produce the most alarming cardiac failure, with pallor, cold extremities, vomiting, and a rapid, feeble heart. In *older children* also severe cardiac disturbance may result. In the worst cases the condition has a striking resemblance to a diphtherial affection of the heart. There are dilatation, feeble tapping heart-sounds, breathlessness, and great prostration. Death in rare instances may occur from syncope. Recovery is, however, the rule, but it is singularly slow, with tendency to relapse, and any imprudence will again produce symptoms of palpitation and discomfort, with some dilatation. In some instances, as Dr. Cheadle has pointed out to me, a harsh basal murmur develops, the exact explanation of which is not apparent.

In *adults*, also, influenza may produce remarkable alterations in the character of the *pulse*. During the febrile stage of the disease this may be unusually *slow*, or after the feverish stage is over there may be *tachycardia*.

There are cases on record in which healthy adults have died from *syncope* after an attack of influenza, and in the elderly with unsound hearts this danger is a very real one during convalescence.

Angina pectoris has many times been directly traced to an attack of influenza. The attacks are somewhat peculiar in their frequency, and they may be severe and fatal. In favourable cases they gradually become less frequent, and eventually after much care and patience there may be complete recovery.

The most frequent injury, however, that results from influenza is a persistent breathlessness and irritability of cardiac action, with pain and distress on exertion, and it is noteworthy that in cases of exophthalmic goitre following influenza the cardiac symptoms are marked, and tachycardia severe.

It cannot be too clearly recognized that influenza may act as a profound cardiac poison, and that over-exertion and strain of the heart when it is thus affected are most dangerous.

A few words are necessary here upon the occurrence of thrombosis in influenza.

Venous thrombosis was frequently noticed in the early phase of the great epidemic, and usually affected the lower extremities. Thrombosis of the cerebral sinuses or cortical veins was also met with. *Arterial thrombosis* with considerable local periarteritis and much pain is a less common occurrence than venous thrombosis.

Prognosis. Influenzal malignant endocarditis is a fatal illness. In the *young and strong* the myocardial affections usually recover completely, but this recovery may be slow, and on this account caution may be rashly dispensed with and the heart consequently overstrained. I have seen a

fatal result follow such imprudent exertion when the heart has been weakened by a recent attack of influenza. Angina pectoris after influenza is dangerous, but the tendency is to slow recovery when every care is taken and the patient is not already suffering from myocardial disease.

In the *elderly*, and those who have already damaged hearts, a severe influenzal cardiac attack is most serious, and may be rapidly fatal, or be followed by a gradual breakdown which is not recovered from.

Treatment. This is in the first place *rest*. Especial care must be taken with elderly patients when they first begin convalescence and leave their beds, and the character of the pulse must then be closely watched. There may be the greatest difficulty in persuading active men of business of the folly of struggling against such warnings as palpitation and breathlessness on exertion in spite of the fact that such symptoms may have been previously unknown to them.

Strychnine (18) and *stimulants* are indicated, and if there has been previous high blood-pressure, nitro-glycerine may be added to the mixture. Later, *exercise* must be gradual and slowly resumed, and in some cases in which the nervous element is prominent, the *Nauheim treatment* (vide Appendix) and exercises advocated by Schott are serviceable. Where dilatation is persistent, this method of treatment may also be useful, and a course of *digitalis*, combined with *bromide of ammonium*, or *iodide of potassium* can be tried.

Sir Lauder Brunton advocates the use of *calcium chloride*, given in doses of from 5 to 10 grains, and prescribed every 4 hours in water to which has been added a minim of the elixir of saccharine.

III. SEPTIC MYOCARDITIS

Acute myocarditis may also occur in association with malignant endocarditis of non-rheumatic origin. The Broadbents, in the fourth edition of their work, figure a very characteristic example, in which large masses of micro-organisms had escaped from the myocardial blood-vessels, and had caused acute inflammatory changes in the connective tissue of the heart, with toxic damage to the muscular fibres. This condition is very difficult to diagnose.

There will be an excited cardiac action and such evidences of cardiac dilatation as have been already given under rheumatism and diphtheria.

In *acute septicaemia* also the cardiac muscle is profoundly damaged. There need not be any definite myocarditis, but hyaline swelling of the muscular fibres as a result of the general toxæmia.

In such cases the impulse may become impalpable, the sounds short, clear, and approximated, and the pulse may for some hours before death be completely lost at the wrist. It is remarkable how in such cases the consciousness may be preserved and the mind be unusually clear.

IV. MYOCARDIAL WEAKNESS ASSOCIATED WITH
PROLONGED PYREXIA

Next to these virulent examples of myocardial poisoning come those conditions of the heart which are met with in prolonged and high fever, from some severe infection such as typhoid fever.

The symptoms are in such cases masked by the primary disease, but the physical signs should be thoroughly recognized.

The *pulse* rises in frequency, and the wave becomes progressively shorter and more ill-sustained. The *impulse* of the heart grows feebler, and there may be some dilatation, though this is not a prominent feature. The *first sound* at the impulse becomes shortened, and the two sounds may for a while be spaced: later they approximate, and eventually a running pulse develops, associated with an almost inaudible first sound and a short, clear second sound.

Treatment. The *treatment* of the heart consists in sparing the patient all exertion by skilled nursing. Most careful dieting is needful to prevent gastric distension, and alcoholic stimulants, gradually increased to meet the progressive failure, are most valuable.

Strychnine, *caffeine* (19), *camphor*, *ammonia*, and *ether* (6), and *adrenalin* in ten- to thirty-minim doses of the adrenalin chloride solution 1-1000, are all useful. *Digitalis* and *strophanthus*, in cases where the digestion is not easily upset, may help to steady the heart, particularly when the action of the heart is rapid, as a result of the continued fever rather than of the actual feebleness of contraction.

The *great point* in the treatment of these cases is to graduate the strength of the remedies to the particular condition, and not to fall into the error on the one hand of unduly stimulating an excited heart, or on the other, of withholding assistance from a labouring one.

B. CHRONIC NON-RHEUMATIC FORMS OF MYOCARDIAL DISEASE

Chronic myocardial disease may result from such causes as :—

1. The healing and scarring of a severe acute or subacute myocarditis, and from disease of the coronary arteries.

2. From continuous and slow absorption of a chemical poison such as alcohol.

3. From prolonged anaemia.

4. Cardiac overstrain.

5. Increased peripheral resistance.

Morbid anatomy. The pathological changes in the myocardium are various:—

1. From the healing and scarring of acute myocardial lesions a more or less diffuse fibrosis will result: the interstitial tissues are increased and sclerosed, there is perivascular fibrosis, and some of the muscular elements that have perished are replaced by this tissue. The changes are usually most apparent immediately beneath the pericardium and endocardium.

When there has been a local inflammatory formation of some considerable size, such as a gumma, the scar will be very evident, and may produce irreparable damage. In the finer cirrheses of the heart there may not be much visible change, but the cardiac wall is unusually tough.

2. There appears some doubt as to whether a sclerosing myocarditis may occur comparable to a sclerosing endocarditis, but the probabilities are in favour of such a condition existing, however exceptional.

3. A fibroid condition of the heart wall has also been frequently noticed in association with sclerosis of the coronary arteries, and by Weigert, Huchard, Lindsay, Steven, and others, is looked upon as a direct outcome of the diminished blood supply.

This result Steven explains as the result of the obliteration of the terminal arterioles of the coronary circulation by the process of arterio-sclerosis. Areas of fatty change in the muscular area supplied by these vessels result, and these damaged areas are replaced by connective tissue hyperplasia.

4. General fatty degeneration of the cardiac muscle is another type of change.

This is also a result of coronary disease, and is in this case explained by Steven as a result of the gradual occlusion of the *main trunks* of the coronary arteries. As a result of this there is a general cardiac anaemia.

The coronary arteries are so often found damaged in fatal cases of myocardial degeneration that a brief account of their lesions forms a necessary introduction to a study of chronic myocardial disease.

Diseases of the Coronary Arteries

These may be divided into two groups:—

I. The result of extension of disease from other parts:

(a) Secondary to aortic diseases.

(b) Secondary to malignant endocarditis with resulting embolism.

a. Acute and chronic aortitis or atheroma may implicate the coronary vessels and partially or completely occlude their orifices, or the same processes by direct extension may spread along the vessels.

b. Embolism of a coronary vessel may be dismissed in a few words. Its cause is the detachment of a fragment of a vegetation from an acute endocarditis and its impaction in the vessel. The usual result is sudden death, but when a small vessel only is obstructed a cardiac infarct is produced from which recovery may occur.

II. Primary disease of the vessels.

1. Acute and chronic arteritis and coronary aneurysm.

2. Endarteritis obliterans.

3. Thrombosis, a result of 1 or 2.

Acute arteritis is doubtless rare, and cannot be diagnosed. Its existence is most obvious when an embolus infects the

arterial wall, and a coronary aneurysm results from the acute inflammation thus produced. Such an aneurysm may be a cause of *sudden death*: there are on record cases of multiple coronary embolism.

Chronic arteritis. Chronic arteritis, giving rise to atheroma or arterio-sclerosis, is the result of the same causes as chronic arteritis in general. Middle age, coupled with a constant high blood-pressure the result of a too liberal diet, is an important factor, especially in those who inherit a tendency to arterial and chronic kidney disease.

Excessive intermittent exertion over many years may also produce aortic atheroma, and in some cases coronary disease.

Chronic bacterial infections are probably also a factor, though this point is not yet thoroughly established. *Lead poisoning*, and *possibly alcoholism*, are included by most writers.

Syphilis may produce disease of the aorta which extends to the coronary vessels, or it may cause primary **Endarteritis obliterans**.

The occurrence of **thrombosis** in the coronary vessels can be readily understood in these conditions, for the flow of blood is impeded, the vessel wall often roughened, and the heart weak.

When thrombosis occurs in terminal arteries a cardiac infarct results. If a large vessel is occluded there is great danger of sudden syncope, or local necrosis may occur, which may later cause a cardiac aneurysm or rupture of the heart.

Morbid anatomy. In atheroma and arterio-sclerosis the vessels are thickened and hard; in some cases there is much calcareous change, and the vessels stand open like rigid tubes.

The *microscopic changes* are at first subendothelial in position, and are either the result of a primary necrosis from deficient nutrition or direct poisoning, or of a primary cellular proliferation with or without subsequent death of the newly-formed tissue. The change may be patchy or uniform, and in advanced cases the intima is destroyed and the muscular tissue of the middle coat also greatly damaged. The diseased areas show a structureless débris.

Endarteritis obliterans is the result of a proliferation of the subendothelial cells, which do not become necrotic, but which may greatly diminish the lumen of the vessel.

I. Chronic arteritis and endarteritis obliterans of the coronary vessels lead to the consideration of two important chronic myocardial affections.

A. The fatty heart. B. The fibroid heart.

A. FATTY HEART

Fatty changes may occur in the heart as a result of other causes than coronary disease ; such are:—

(a) Bacterial poisons ; for example, those of diphtheria and influenza, rheumatism, and beri-beri.

(b) Chemical poisons, such as phosphorus, arsenic, and alcohol.

(c) Altered blood states, such as pernicious anaemia.

Nevertheless, important though these are, they do not give the picture of the clinical disease 'fatty heart', which results from coronary disease.

This disease is more frequent in males than females, and generally commences in late middle life.

Morbid anatomy. The heart may or may not be much dilated, and there is usually a certain degree of associated fibroid change.

The muscle is sometimes greatly altered even to the naked eye. It is pale, streaky (tabby cat striation), and on section the knife is covered with oil globules. The muscle substance is exceedingly soft and greasy, and occasionally in the right ventricle there may not be any detectable muscular fibre.

Under the microscope some of the fibres may be unrecognizable, and in others the transverse striation is lost, and large drops of fat are visible within the fibre.

This process of *fatty change* has been long looked upon as a different one to that of *fatty infiltration*, but the recent work of Rosenfeld tends to show that the distinction between them is not perhaps so great as was thought. Dudgeon's still more recent investigations lead one, however, to believe that fatty degeneration or change is not a passive deposit, but a chemical process in the muscle fibre itself. This observer points out that 10 per cent. of the total solids of normal cardiac muscle consist of fat, which cannot be demonstrated histologically, and he suggests that so far as diphtheria is concerned, the poison liberates this fat from the complex combination in which it is bound in health, and when thus liberated it can be stained by the ordinary histological methods.

He also points out that the amount of fat in the heart muscle may be actually increased in some cases of fatty changes, and believes this to be due to imported fat, which the toxic processes have prevented from being duly oxygenated. This added fat is accordingly of the nature of a fatty infiltration.

Symptomatology. The symptoms are vague, and this is one of the forms of heart disease in which *sudden death* may be the first indication of anything amiss.

In a considerable number of cases there are, however,

suspicious symptoms which may lead to a diagnosis. The *facial aspect* may be pale and waxy, although, on the other hand, it may be particularly young and fresh. The *breath may be short* on moderate exertion, but still more suggestive, especially when occurring in a stout middle-aged man, are the occurrence of *syncopal*, *epileptiform*, or *apoplectiform attacks*.

The *extremities* are often cold, and there is a tendency to *sleep heavily after meals*, or at various times during the day. *Digestion* is slow, and there may be general *lassitude*.

The syncopal attacks often result from slight causes, such as digestive disturbances, sudden emotions, or accidents; they may be of long duration, and sometimes most severe. The epileptiform attacks are associated with a slow pulse, and their pathology is considered later under the Stokes-Adams syndrome, Chapter xiv, p. 249.

The apoplectiform seizures resemble those that occur in cerebral haemorrhage.

Lastly *angina pectoris* may occur in association with a fatty heart; vide Chapter xiv, p. 289.

The physical signs. 1. *The pulse.* The wave is short and ill-sustained, and sometimes unusually slow. The rhythm may be regular or irregular.

2. *The heart.* The impulse is not visible and may not be palpable, the area of dulness may not be increased, the heart sounds are short and clear. Sir W. Broadbent points out that if the patient is made to walk briskly and the heart again examined, it will be found that it has failed to respond to this exertion, and is accordingly more rapid and irregular in its action.

The **diagnosis** is generally a matter of conjecture unless most of the symptoms are present, and the disease far advanced, but this conjecture is sufficiently probable to deter-

mine measures to be taken for treatment, and is founded upon these points:—*A poor circulation with slight cyanosis of the extremities. A heart somewhat enlarged, with feeble irregular sounds. A patient past middle life.*

The prognosis under such circumstances is very grave, and the duration of life necessarily most uncertain. A sudden shock may prove instantly fatal, while on the other hand, if a quiet and peaceful life is possible, the end may be postponed for some years.

B. FIBROID HEART

In addition to coronary disease the following are probable causes of fibroid change in the myocardium:—

1. Subacute myocarditis, syphilitic, rheumatic, &c.
2. Gummatous deposit.
3. Chronic venous congestion from prolonged pulmonary disease.

Symptomatology. The symptoms are chronic in their course and slow in their development. The patient is usually a man of middle age, and comes under treatment for *progressive shortness of breath on exertion*. There are *cyanosis* especially on exertion, and some *puffiness of the ankles* at night. In association there are often well-marked signs of *arterial degeneration* and *emphysema*.

Physical signs. The *pulse* is irregular, though not invariably so, and may be easily compressible, although the arterial wall is thickened.

The *impulse of the heart* is displaced downward and outward, is usually not powerful, and may be entirely concealed by the emphysematous lungs. The *cardiac area* is enlarged both to the right and left. The *first sound* is muffled and prolonged, and followed by a *faint systolic murmur*. At the base the first sound may be inaudible. As the case pro-

PLATE XIV



A

Section through part of the left ventricle, showing interstitial fibrosis, the result of chronic myocarditis. *By permission of Messrs. Lippincott.*



B

Section through part of the wall of the left ventricle, showing perivascular fibrosis, the result of chronic myocarditis. *By permission of Messrs. Lippincott.*

gresses all the signs of failing heart may develop, including *Cheyne-Stokes respiration*, *apoplectiform* and *epileptiform seizures* and *syncopal attacks*.

The diagnosis cannot be made with certainty, and the prognosis is necessarily grave.

C. FATTY INFILTRATION OF THE HEART

Less serious than fatty heart, but closely allied in some respects, and most conveniently considered here is *fatty infiltration of the heart*.

There is a great increase in the fat which is naturally present, and with it an infiltration of the muscular substance of the heart wall, particularly on the right side.

There is a decided family tendency to adiposity, but in many cases this condition is much encouraged by a good appetite and digestion, unbalanced by sufficient exercise and elimination.

The Symptoms are *general stoutness* with *feeble circulation*. Exertion is irksome, and accompanied by *breathlessness* and *slight cyanosis*. The breathing is embarrassed.

Physical signs. The pulse is soft, and the pressure low. Examination of the heart shows no striking change. The impulse is feeble, the cardiac dulness pushed slightly upward, and the sounds are faint.

This condition can be much improved by suitable treatment.

D. SYPHILITIC DISEASE OF THE HEART

Syphilis produces great changes in the arteries, and may sometimes also damage the heart severely, attacking the valves, myocardium or pericardium.

1. Valvular lesions. These are usually subacute or chronic, and affect the aortic valve rather than the mitral.

One is naturally not on very firm ground when considering syphilitic endocarditis, for the cause of the disease is not yet definitely ascertained, the fate of the *spirochaeta pallida* being still in the field of controversy. There is, however, good clinical evidence pointing to a *syphilitic aortitis*, a process which may also involve the *aortic valves*, and then produce considerable incompetence.

There is nothing unusual in the aortic regurgitation that results, but the heart does not compensate so well as in rheumatism, for the disease appears in adult life and is complicated by arterial degeneration, and sometimes by disease of the wall of the heart. There may be severe attacks of *angina pectoris* in these cases, attributed by some to aortic disease, and by others to the implication of the coronary vessels.

2. *Syphilitic affections of the myocardium* are of very great practical importance. They are sufficiently infrequent to escape the memory, and their course is often so latent as to baffle diagnosis, yet they are a cause of *sudden death* in the very prime of life.

The nature of the changes are either *gummatous* or there is a more *diffuse fibrosis*. The *ventricles*, more particularly the left one, are more often affected than the auricles. The gummata vary in size from a small pea to a considerable tumour, which produces around it a swelling of the myocardium that gives a false impression of hypertrophy. These gummata may be softened in their centre, or may have organized into a tough scar. Favourite situations are the *apex of the left ventricle* and the *interventricular septum*; they may also be found in the right ventricle. We are indebted to Sir Samuel Wilks for the pioneer work upon this subject in England, and S. Phillips, in 1897, gave a valuable clinical account of some striking examples of this affection. Accord-

ing to his observations the great danger lies in the occurrence of *gummata in the left ventricle*.

Symptomatology. If the *gummata* are very minute they may give rise to no symptoms at all, but otherwise they usually produce *dyspnoea, angina pectoris, precordial discomfort and palpitation*.

Physical signs. The *heart* may show no enlargement, but the *pulse* becomes rapid, excited, and sometimes very irregular. Other grave symptoms that may result are syncopal attacks, epileptiform seizures, and finally sudden death.

As a result of a softening *gumma* a *cardiac aneurysm* may form, and *rupture of the heart* occur.

Myocardial fibrosis is most difficult of diagnosis. A prominent symptom is *dyspnoea* with loss of strength and a feeling of great weakness, dilatation of the heart occurs, and if the right ventricle is implicated, tricuspid regurgitation supervenes.

The **diagnosis** of cardiac syphilis is difficult, but should always come to mind when a young adult, who has not suffered from rheumatism, but has contracted syphilis, presents symptoms of heart disease. In such case particular stress must be laid upon the occurrence of *angina pectoris* and a rapid irregular pulse, with a heart that shows no enlargement.

The **prognosis** is serious. Doubtless small *gummata*, if they are treated in the early stage, are cured, but in many cases the nature of the case is not suspected, or fatal symptoms occur without warning. Fibrosis of the myocardium is incurable, whether it be from syphilitic disease of the heart wall, or the result of endarteritis obliterans of the coronary vessels.

The **treatment** has clear indications. Absolute rest, and the exhibition of mercury and potassium iodide until

all the symptoms are controlled, are the chief factors for success.

E. THE HEART IN ALCOHOLISM

Acute and chronic alcoholism seriously damage the heart.

After death from *delirium tremens* the cardiac muscle is usually pale, flabby, and shows fatty change. In the more chronic forms there is usually an excess of *epicardial fat*, the cavities are *dilated* and the walls often *hypertrophied*, and the organ very large and heavy.

The *myocardium* sometimes shows much disease. The muscular bundles are separated by fatty infiltration, and the fibres may show great fatty degeneration. In other cases there is but little obvious change.

In addition, alcohol is probably a factor in *arterio-sclerosis*, and when the first part of the aorta is affected the aortic valve may also become involved.

The *symptoms* may be acute or chronic. In acute cases, associated with *delirium tremens*, there are *livid pallor* and *coldness of the extremities*.

The *pulse* is small, often irregular, frequent, and even running in character.

The *heart* may or may not be dilated, but the tendency in any case is for the first sound at the impulse to become short and faint, and the pulmonary second sound to be accentuated. In chronic cases the *pulse* is rapid and feeble, and in the chronic cases with dilatation often very irregular. The physical signs are generally those of a dilated and hypertrophied *heart* with breathlessness and sometimes extreme oedema which is unusually firm. This condition in hospital practice often complicates chronic valvular disease in adults, and is a factor very detrimental to recovery. In my experience the importance of chronic alcoholism—not

necessarily drunkenness—as a cause of dilatation and hypertrophy of the heart, of myocardial weakness, and sudden death in those with organic valvular disease is hardly sufficiently realized by students.

F. THE HEART IN ANAEMIA

In all forms of *anaemia* the tendency is for the myocardium to undergo fatty change. This is a most striking feature in *pernicious anaemia*, in which, in addition to profound alterations in the walls of the ventricles, the papillary muscles are severely damaged, and fatty changes are also met with in both the large and small arteries. In *chlorosis* the same condition is met with to a lesser degree, but the disease is so much more frequent that the circulatory conditions in this affection will be used in illustration of this group.

The **symptoms** are *breathlessness, palpitation, pain over the heart, and syncopal attacks*. These by no means always bear a direct relation to the degree of anaemia.

The **physical signs**. The *pulse* is frequent, variable in force, but the wave is by no means always easily compressible.

The impulse of the *heart* is irritable, and the area of the cardiac dulness is frequently increased both to the right and left.

Haemic or functional bruits are often present, and sometimes are audible at all four orifices, the *bruit de diable* is another common physical sign. Some *oedema* of the lower extremities may be present.

The chief difficulties in the **diagnosis** of this condition are the separation of chlorosis from a *secondary rheumatic anaemia* with organic heart disease. I have found that the abrupt first sound of the heart in a nervous chlorotic girl is not uncommonly mistaken for evidence of mitral stenosis.

In these cases the history must be carefully reviewed, the effect of posture upon the true character of the murmurs studied, the blood examined, and other symptoms of chlorosis or rheumatism looked for.

Anaemia with renal disease in young women is another pitfall. Much emphasis must be laid in renal disease upon albuminuria with casts in the urine, arterial hypertonus, and evidence of cardiac hypertrophy.

Lastly, chlorosis may be complicated by *Influenza* or by *overstrain* of the heart, and then serious symptoms of dilatation result. When anaemia is *complicated by gastric ulcer* of sufficient severity to necessitate the employment of rectal feeding, this condition of the myocardium should be borne in mind, and every precaution taken to prevent fatal syncope.

The **prognosis** is usually good, but if the anaemia has been of some duration, and particularly if the heart has been strained by over-exertion, these cases may be most obstinate.

G. STRAIN OF THE HEART

This is a condition of much practical importance and not infrequent. The over-zealous pursuit of athletics from youth to manhood accounts for a certain number of these cases, and since girls and women have joined in such vigorous pastimes, heart strain has also appeared in this sex. There can be no question as to the value of athletic exercises, and it is rather the insatiable appetite of the 'mad English' for such amusements, than the amusements themselves, that are to blame. The well-known writings of Myers and da Costa have brought into prominence a serious form of heart strain among young soldiers, the result of prolonged exertion when heavily accoutred, and this form will be considered first.

1. Soldier's Heart

a. Symptomatology. Among the chief symptoms are: *palpitation, breathlessness, and faintness on exertion.* There are often also *tremor* and *great nervousness.* A very prominent feature is the disappearance of these symptoms upon rest and their obstinate reappearance on exertion.

b. The physical signs. The physical signs are definite: when at rest the *pulse* is regular, though possibly of small size and too easily compressible. On exertion, however, the frequency runs up to 110-140, and the beats become irregular. The impulse of the *heart* on exertion is forcible and tumultuous, and there may be considerable dilatation, although this is by no means constant. The auscultatory signs are not remarkable; the sounds may be unusually loud and sometimes there is a puffing mitral systolic murmur, but neither is constant. There is no dropsy, enlargement of the liver, or cyanosis.

2. Milder Forms of Heart Strain

The heart strain from athletic over-exertion is as a rule milder in type and more transient in duration. It has been pointed out by writers on the subject that among soldiers the patients are often mere youths, of ordinary, if not poor, physique; that their respiratory movements have been hampered by improper clothing and the weight of accoutrements, and that, lastly, they have been damaged by tropical or other diseases, or by the abuse of alcohol and tobacco.

In the second group of cases which cannot be rigidly separated from the preceding there are also very often *secondary factors* which are of great importance in producing the breakdown, and have close bearing upon treatment.

The precise explanation of this form of heart affection is difficult, because of the complexity of the factors included.

Physiological investigation teaches that sudden severe strain does for a short time greatly *increase the peripheral resistance*, and this must clearly throw a strain upon the left side of the heart. All athletes who have studied medicine will also admit that there may be an equally great if not greater strain upon the right side of the heart from the tendency to get *over distension of the right ventricle*. I look upon *imperfect training* as the most important factor in this form of heart strain. The athlete who from necessity has been compelled to leave his pursuits for active brain work will, or should, realize that it requires some years of steady training to school his brain to continuous and efficient mental effort. It is so also with athletics. In youth the need for training is at a minimum, but as adult life is reached the need becomes more apparent. There are many important results obtained by ordinary training. *Harmony between eye and muscle* is one result, and another of special importance is *co-ordination* between the *cardiac, respiratory, and muscular movements*. The first saves the athlete in severe games a great amount of exertion, and preserves him from undue and sudden muscular strain; the second protects him from over-distension of the right side of the heart. Again, in ordinary training we approximately know our strength, whereas out of training we are apt to remember our best days and to think we are still living in them. Young men and boys in training may strain their endurance past the level of discretion, and show symptoms of urgent distress, yet no permanent harm is done. This is partly because of their great recuperative power, and because also the strain, though extreme, falls upon sound and practised tissue. It is a very different matter if such a strain is put upon the heart of an individual who has suffered recently from severe influenza. From this I have seen severe tachycardia in a young man, who had

previously been in good health result fatally on the fifth day. It is again a very different matter when the subject is a *middle-aged man* out of condition ; such an exertion may be, and often has been, the commencement of a fatal illness.

Thus it becomes apparent that exertion, even when very severe, does little or no harm to the healthy and trained tissues, and that when injury does follow there is often some secondary factor at work. Acute *illnesses* partially overcome, actual *myocardial diseases*, or constitutions damaged by *faulty habits* may be responsible, and in young women *anaemia* must be remembered.

There is yet another factor which is superadded, and which complicates the practical side of this subject, and this is the *nervous element*. In young women this may be very prominent, and a great many athletic men, I would add, are also highly nervous. In such cases as these some damage has been done to the heart, and cardiac discomfort arisen as a result, but this is rendered much more acute and severe by a high degree of nervous disturbance.

The symptoms differ considerably.

In the acutely severe cases there are *dyspnoea*, *lividity*, and *exhaustion* at the time, and afterwards, on exertion there develop the *rapid irregular pulse* and *tumultuous heart* described above. When cases such as these progress unfavourably the case drifts gradually into one of chronic dilatation of the heart (q. v., p. 220).

The milder cases may only show an irritability of the heart brought on by slight exertion, emotion, or smoking.

The prognosis in the more severe cases is not good, the condition relapsing on active exertion ; the milder cases generally recover, although slowly.

CHAPTER XIII

MYOCARDIAL AFFECTIONS (NON-RHEUMATIC)

(continued)

The heart in renal disease—Pathology—Symptoms—Hypertrophy of the heart—Causation of—Symptoms—Prognosis—Treatment—Dilatation of the heart—Symptoms—Diagnosis—Prognosis—Pericardial affections in renal disease—Symptoms—Diagnosis—Prognosis—Treatment—Myocardial affections, the result of pulmonary disease—Causation and results—Treatment of chronic myocardial affections—General measures in the milder forms—Toxic cases—Anaemic cases—Cases of fatty infiltration—The graver forms—Severe heart strain—Fatty and fibroid heart—Renal dilatation—Cardiac dropsy—The Nauheim treatment.

MYOCARDIAL disease may be a result of high peripheral resistance, and this resistance may be in the systemic circulation as in renal disease, or in the pulmonary circulation as in chronic emphysema and bronchitis.

H. THE HEART IN RENAL DISEASE

Since Bright demonstrated the important relation that exists between kidney disease and hypertrophy of the heart, the frequency of cardiac lesions in renal affections has become thoroughly recognized.

These lesions must be distinguished from those in which the heart disease is primary and has produced chronic venous congestion of the kidneys. Such a condition no doubt in time adds a renal factor to an old-standing heart disease, but in the class of case considered here, one of two things has happened :

either the heart and the kidney have been both damaged by some poison, microbic or otherwise, or the kidney has been attacked, and then as a direct result of the renal disease the heart has suffered.

The first group, in which both kidney and heart have been affected, is a very interesting one of which there is more to be learnt.

1. Combined Cardiac and Renal Disease

Newton Pitt collected a series of cases in which mitral stenosis was associated with chronic renal disease, and in which one suspects a chronic renal infection running hand in hand with a chronic cardiac infection.

In scarlet fever, in rheumatic fever, and in some cases of malignant endocarditis, active endocarditis and acute nephritis may occur together quite irrespective of embolism.

It is sufficient to mention this group, for the clinical signs are those of endocarditis and nephritis and do not need further description.

2. Cardiac Disease, the Result of Renal Disease

In all forms of renal inflammation the heart suffers to some extent, but there is much dispute as to the exact explanation of the way in which these changes are brought about, and it seems very possible that no one explanation will suffice for all cases. The two cardinal lesions that result are *dilatation* and *hypertrophy*, and it is in renal disease that we meet with striking examples of hypertrophy and dilatation in the entire absence of endocarditis or pericarditis. In this chapter accordingly these two conditions of hypertrophy and dilatation will be considered in detail.

It is the *left ventricle* which particularly hypertrophies in renal diseases, and in many cases this part of the heart alone. Nevertheless, it is equally certain that *sometimes the right*

ventricle is also implicated, a fact which has to be taken into account in all explanations of the causation of hypertrophy in kidney disease.

In the *contracted forms of kidney*, whether primary or arterio-sclerotic, the left ventricle is sometimes enormously hypertrophied and the right side of normal thickness. In these cases *dilatation is a terminal phase*, an evidence of the failure of the heart to cope with the great peripheral resistance. In *acute and subacute nephritis* on the other hand, the more rapid accumulation of irritant nitrogenous substances in the blood may raise the peripheral resistance so rapidly that there results a *primary dilatation*, which may never be recovered from, or which may be followed by hypertrophy, and finally by the dilatation of complete failure.

The nitrogenous bodies retained in the system irritate the musculature of the entire cardio-vascular system, but the strain so far as the heart is concerned will be felt much more severely by the left side, because the hypertonus produced in the arterioles of the systemic circulation causes a vastly greater effect upon the systemic peripheral resistance than a similar occurrence in the pulmonary circulation. There are accordingly two factors tending to cause hypertrophy of the left ventricle: the direct irritant effect of the nitrogenous poisons upon the cardiac muscle, and the high peripheral resistance produced by their irritation of the muscular walls of the systemic arteries. It is possible also that these poisons damage the capillaries or tissues in their immediate vicinity, in such a way as to raise the peripheral resistance. At the bedside it is possible to trace in the primary renal affections two forms of this increased peripheral resistance, the first a *persistent one* dependent upon permanent change, the result of chronic irritation, and the second a *fluctuating spasm* of the vessels, which may

in a few hours send up the blood-pressure with great rapidity and make the radial arteries feel like whip-cord. During one of these storms there may be acute heart failure, convulsions, haemorrhages from the colon, or under the skin.

It would appear that when *dropsy* occurs in the acute and subacute cases of renal disease, the nitrogenous retention products are drained into this fluid and the peripheral resistance is to some extent eased by this cleansing of the blood. In such cases the hypertrophy of the heart may not be great, and if the disease rapidly subsides there may be complete recovery, an occurrence by no means rare in childhood.

In the *arterio-sclerotic* form of contracted kidney, the disease commences in the blood vessels, and later, through implication of the renal arterioles, affects the kidney. In such cases the poison of gout, lead, and possible microbic infections, produces this *primary arterio-sclerosis*, which increases the strain upon the left side of the heart by raising the peripheral resistance. In response to this extra amount of work, the left ventricle slowly and steadily hypertrophies.

Symptomatology. *In Acute and Subacute Nephritis.* The prominent symptoms are usually those of renal disease, but should the heart dilate there is *breathlessness*, and even *fatal syncope* has been recorded by Goodhart. The *pulse* is increased in rate, easily compressible, and sometimes irregular. The cardiac *impulse* is diffuse and the area of *cardiac dullness* increased to the left.

The *first sound* is prolonged and the rhythm of the heart tic-tac; if the condition is more serious the first sound becomes shortened, and there may be a cantering rhythm due to reduplication of the first and second sounds.

When the heart rallies the pulse improves and the wave gradually alters in character, becoming now less compressible than normal. Frequently there is also hypertonus of the arterial wall.

The heart's impulse becomes forcible, and the area of precordial dulness diminishes with the recovery from the dilatation. The first sound over the impulse is low-pitched and the aortic second at the base intensified.

In *chronic renal disease* the gradual hypertrophy rarely gives rise to any symptoms, and it is as a rule the commencement of a *terminal dilatation* which first brings the patient for advice about the heart, although in some cases persistent insomnia and a feeling of tension in the head, with occasional epistaxis, may lead to the discovery.

A. HYPERTROPHY OF THE HEART

There are many causes of hypertrophy of the heart, and this will be a convenient opportunity to classify them and give a brief description of the condition.

1. Intra-cardiac Causes

1. Valvular lesions, e. g. aortic regurgitation and stenosis. Mitral regurgitation and stenosis. Tricuspid regurgitation and stenosis. Pulmonary regurgitation and stenosis.

2. Pericardial lesions. Some cases of adherent pericardium.

2. Extra-cardiac Causes

1. Physiological hypertrophy due to increased functional activity, the result of prolonged muscular exercise.

2. Hypertrophy, the result of persistent high peripheral pressure from renal disease or primary arterio-sclerosis and atheroma.

3. Hypertrophy, the result of chronic pulmonary diseases, such as emphysema, asthma, and repeated bronchitis.

4. Hypertrophy, the result of pathological stimulation by excess of thyroid secretion or other poisons causing persistent increase in the rate of the heart's action.

Some of these causes affect the left ventricle in particular, others the right.

Thus:—

Aortic disease, Renal disease, Arterio-sclerosis,	} affect the left ventricle particularly.	Mitral stenosis,	} affect the right ven- tricle partic- ularly.
		Lesions of the	
		pulmonary	
		valve, Obstructive pulmonary disease,	

Symptomatology. The symptoms are few. There may be *distress* from the violent pulsation or from palpitation, and sometimes *insomnia* of the active type is troublesome. In some cases there is *dyspnoea*.

The Physical signs. The *pulse* will depend upon the cause. In renal disease the wave is prolonged, rises gradually, and is difficult to compress. The vessel is felt between the beats and the arterial wall is thickened.

The impulse of the heart is displaced downward and outward into the sixth or seventh space, and is heaving in character. Percussion shows an increase in the cardiac dulness to the left and downwards. The first sound is prolonged, and low-pitched, and the aortic second sound may be accentuated and reduplicated.

The Diagnosis. Although from these physical signs it would appear to be easy to diagnose hypertrophy of the left ventricle, it may be most difficult, because there is frequently in both chronic renal inflammation and arterio-sclerosis

marked emphysema which masks the cardiac dulness and conceals the forcible impulse.

The **Prognosis** in hypertrophy depends entirely upon the cause. Whatever this may be there are certain valuable signs which are of importance as warnings of *commencing dilatation*. These are:—

1. Breathlessness on exertion.
2. Diminution in the heaving character of the impulse.
3. Prolongation of the first sound, and tic-tac heart sounds.
4. Reduplication of the first sound at the apex and of the second sound at the base.

There is no **treatment** for hypertrophy—in renal disease the treatment must be directed to the kidneys and to the peripheral resistance. By eliminants and evacuants, coupled with nitrites, and by correction of the habits as to diet and exercise, the blood-pressure may be kept in some degree under control, and thus indirectly the hypertrophy of the heart also.

B. DILATATION OF THE HEART

Dilatation of the heart is a much more serious condition. The many causes may be conveniently grouped thus:—

Primary dilatation.

1. Causes due to damage of the myocardium by bacterial infections, e.g. diphtheria, rheumatism, influenza.
2. Due to chemical poisons, for example, alcohol, tobacco, overdoses of salicylates, stramonium, and lobelia.
3. Due to mental emotion and shock, and excesses.
4. Due to physical over-exertion, particularly when out of training or ill-fed.

Secondary dilatation.

1. The result of valvular disease.
2. Of adherent pericardium.

3. Increased peripheral resistance as in renal disease and arterio-sclerosis.

4. Secondary to disease of the coronary vessels.

5. The result of various forms of anaemia.

6. The result of persistent tachycardia.

These two divisions overlap one another, as for example, when over-exertion produces dilatation in a heart already damaged by degenerative changes, the result of disease or age.

From a survey of these causes, it will be apparent that various types of dilatation are met with in heart disease.

1. Simple dilatation—from direct damage to the muscle of the heart, or from acute strain of the heart.

2. Compensatory dilatation—as in aortic regurgitation, by which the increased quantity of blood in the left ventricle at the end of diastole, is accommodated.

3. Dilatation with hypertrophy—as in compensated aortic regurgitation ; where the dilatation again is compensatory.

4. Hypertrophy with dilatation—when the dilatation is due to failure, as in the failing heart of many valvular lesions.

With progressive dilatation, the systole of the ventricles becomes less effectual and the residual blood after the ventricular contraction increases gradually in amount.

Renal disease or overstrain of a heart degenerated by chronic alcoholism, both furnish very characteristic examples of *chronic dilatation*. In the renal cases the left ventricle suffers disproportionately.

DILATATION IN RENAL DISEASE

Symptomatology. Dilatation of the heart in renal disease commences very gradually, and the symptoms can be grouped into :—

1. The general.

2. The special.

1. The general symptoms are the result of the diminishing power of the general circulation, and show themselves by:—

(1) Dyspepsia, flatulence, and irregularity of the bowels.

(2) Lack of mental power, nervousness, disturbed sleep, and melancholy.

(3) Cold extremities.

2. The special symptoms are, among others:—

(1) Breathlessness on slight exertion, cardiac asthma, and palpitation.

(2) Oedema of the ankles and feet, especially at night.

(3) Cyanosis.

As the disease advances, all the symptoms increase in severity. *Sleeplessness* becomes aggravated, and sleep made miserable by horrible dreams. The patient may be unable to lie down at all in bed, owing to the distressing sense of suffocation. The eyes are suffused, the cheeks purple and bloated, and the hands cold. *Oedema* increases, and may be complicated with pleural and peritoneal effusions which make the patient helpless, and add to the general circulatory embarrassment. *Little urine* is passed, and this is loaded with urates, and albuminous. There may be complete *loathing of food*, and much distress from flatulence and constipation.

The Physical signs. The *pulse* is frequent and irregular in force and rhythm. The wave may be large or small, but in both cases it is easily compressible and not felt between the beats. All the characters are brought out by slight exertion.

Examination of *the heart* shows displacement of the impulse outward, or downward and outward. It may not be visible, or there is a wide area of diffuse pulsation. On palpation a tapping beat is detected, with which there may also be felt

a faint sensation of a heaving left ventricle, the result of previous hypertrophy.

The most important auscultatory sign is a *short, sharp, first sound* which may or may not be accompanied by a systolic murmur, soft in character.

The interval between the cardiac sounds is at first increased, and later diminished.

The *secondary results* of the cardiac weakness are shown in all the important organs.

The stomach is dilated, and there is a catarrhal condition of this organ and the intestines. The base of the lungs are engorged and there is frequently bronchitis, infarction, and pleural effusion. The jugular veins are distended and towards the end irregular, and Cheyne-Stokes breathing may supervene, or cerebral softening from arterial thrombosis occur.

The Diagnosis. Apart from mistaking for dilatation of the heart an alteration in its relation to the chest such as occurs in thoracic deformities or from retraction of a lung, the two great difficulties are:—

(1) Pericardial effusion; and

(2) Some cases of mitral stenosis in which the presystolic murmur is absent or has disappeared.

The differential diagnosis of a pericardial effusion has already been considered under suppurative pericarditis (p. 172).

In mitral stenosis there is often a history of rheumatism, and on the other hand no obvious renal or other cause for dilatation. The first sound in mitral stenosis is peculiarly short and sharp, and very careful auscultation may detect a slight presystolic murmur, which is sometimes strictly localized.

When dilatation of the heart has been diagnosed, the next step is to discover its cause.

The family and personal history is inquired into, and the presence or absence of valvular disease or pericardial adhesion determined. The cause of the breakdown and the age of the patient are of importance. If the condition is primarily renal, there may be evidence of previous high peripheral resistance in the subdued heaving of the chest, and the thickened arteries. Chronic asthma and the uraemic odour of the breath may also attract attention. If there is some recovery under treatment, the specific gravity of the urine may fall to and keep under 1010, suggesting chronic interstitial nephritis or the arterio-sclerotic kidney, and in other cases renal casts may be present continually. The retinae should always be examined for renal retinitis.

Prognosis. The chief factors in making a prognosis are :—

- (1) The urgency of the symptoms.
- (2) The degree of dilatation.
- (3) The cause of the breakdown.
- (4) The age, habits, and family history of the patient.

A rapid, feeble pulse, a short interval between the two cardiac sounds, an impalpable impulse and a large precordial dulness signify a grave degree of dilatation.

The outlook is the more serious when the breakdown has been stealthy, or the exciting cause a slight one. A previous history of alcoholism, a family history of renal disease or heart disease are also unfavourable.

PERICARDIAL LESIONS IN RENAL DISEASE

The last lesions to be considered in renal disease are the pericardial. These can be conveniently classified into :—

1. Hydrops pericardii.
2. Acute pericarditis.

a. Due to a simultaneous infection of the pericardium and kidney.

b. As a terminal event in chronic renal disease.

1. **Hydrops pericardii.** This is a serous effusion of a dropsical nature into the pericardial sac. As compared with the pleural and peritoneal effusions it is rare. Unless there is some additional inflammatory element the effusion seldom reaches a pint in amount.

Symptomatology. The condition is remarkably latent, but may cause considerable *dyspnoea* or intensify it if already present. The signs are those of pericardial effusion which have been detailed in Chapter x, p. 170.

2. (*a*) **Acute pericarditis** may result from a rheumatic or scarlatinal infection of the heart and kidneys; or, again, from a pneumococcal or septic invasion of both organs.

(*b*) In the *last phases* of chronic renal disease pericarditis may also develop and prove rapidly fatal.

Whether this is the direct result of profound uraemic poisoning, or a terminal infection in tissues of little resisting power, it is difficult to determine.

The Diagnosis. The particular point to be remembered in the diagnosis of pericarditis in renal disease is the multiplicity of causes for *dyspnoea* in kidney affections. These are:—

(1) Respiratory, e. g. pleural effusion, bronchitis, oedema of the lungs and oedema of the larynx.

(2) Nervous—uraemic asthma.

(3) Cardiac—dilatation, pericarditis, and hydrops pericardii.

It is, then, essential in renal *dyspnoea* to investigate carefully these various organs before deciding upon the causation, and the method of treatment.

MYOCARDIAL AFFECTIONS DUE TO INCREASED PERIPHERAL
RESISTANCE IN THE PULMONARY CIRCUIT

These may result from :—

(1) Failure of the left side of the heart, the result of valvular disease.

(2) Failure of the left side from dilatation.

(3) General cardiac dilatation.

(4) Disease of the pulmonary valve.

(5) Chronic pulmonary disease.

The result is *tricuspid regurgitation*. This has already been described under chronic valvular disease. Where weakness of the right ventricle is the prominent feature in a case of cardiac disease, Sir W. Broadbent has emphasized the importance of *syncope* rather than breathlessness as a feature of the symptoms.

THE TREATMENT OF CHRONIC MYOCARDIAL AFFECTIONS

A. Mild Forms

Among the milder forms of myocardial affections are included the less severe toxic cases, the chlorotic, cases of fatty infiltration, and the milder forms of heart strain.

General measures. (1) For the toxic cases, fresh air, regular exercise, ample but digestible food, and quiet, regular habits with the avoidance of tobacco, are indicated. Some alcohol with the meals need not be forbidden.

A prescription of *iron* and *quinine* (21) and *strychnine* is useful. In some post-influenzal cases which have become obstinate the *Nauheim treatment* is of value.

(2) Fresh air, rest, and sunshine are indicated in *anaemic* cases. If there are considerable palpitation and breathlessness, *digitalis* may be added to a mixture of *iron* and *arsenic*,

and the *bowels* should be regulated by saline or (8) an aloetic aperient.

(3) In cases of *obesity* with fatty infiltration it is often difficult to get the patient to co-operate in the treatment. The indications are nevertheless clear.

Daily exercise in the open air is essential.

The diet should be chiefly nitrogenous (Appendix, diet 3), and a sparing amount of fluid permitted only with the meals. The necessary amount is made good by slowly sipping hot water midway between the meals.

The bowels are regulated, and an occasional *mercurial* and *rhubarb* pill given if there is plethora. A cardiac tonic is helpful at the commencement of treatment.

In many cases the regimen is more effectually carried out at a Spa.

B. Severe Forms

Among the graver forms of chronic myocardial affections are the more severe forms of heart strain, the fatty and fibroid heart, the heart of advanced renal disease, alcoholism, and obstructive pulmonary disease. In some of these conditions there may be extreme dilatation and great dropsy.

SEVERE HEART STRAIN

This is rebellious to treatment.

Rest is indicated while the symptoms are urgent, and if possible the heart should gradually be trained back to efficiency by graduated exercise.

General and cardiac tonics are indicated, and if there is much nervousness; the *bromide* (14) and *valerianate of ammonia* are useful. It may be found that all attempts at exercise, whether active or passive, are harmful, for there is sometimes an extraordinary inability on the part of the heart to respond to physical movement. In such cases entire rest

is necessary. This difficulty warns us to order exercises with caution, to watch their effects closely at first, and to make no rash promises as to their efficacy.

II. FATTY AND FIBROID HEART

If the condition is suspected early, the treatment consists firstly, in advising as to daily exercise. Walking, riding, gentle cycling and golf are allowed, but it is important not to overtire the heart.

Tepid baths should be used. Resistance movements are useful. The diet should be as advised for fatty infiltration. The tonics of most value are—*iron* (21), *strychnine*, *arsenic* (25), and *sodium formate* (15). The bowels must be regulated.

When the condition is more advanced, exercise should be if possible persisted in, but should be confined to walking on the flat. High staircases are most harmful.

Resistance movements are usually contra-indicated, and particular attention must be given to the management of the digestion. A short rest should be taken before lunch, and a longer before dinner. Unless there is reason to the contrary, stimulants are permitted with the meals. Extremes of cold and heat are detrimental: warm clothing and a warm winter climate are advisable.

A prescription containing *strychnine* and *strophanthus* may be given, and a *carminative mixture* (6) should be at hand in cases liable to syncopal attacks.

In the most severe cases *digitalis* and *strophanthus* are not devoid of risk, and the treatment must be to a great extent palliative. Care of the digestion and regulation of the bowels stand first. In the apoplectiform attacks stertor can be averted by turning the patient on the side, and Cheyne-Stokes respiration relieved by *oxygen inhalations* and *strychnine*.

Anginal attacks must be treated by the indications given under angina pectoris for restlessness and sleeplessness. The *bromides* are preferable to opium, and *trional* and *sulphonal* may bring relief. If these fail *opium* should be tried, but cautiously, and preferably by the mouth. *Chloral* is contra-indicated. Yeo advocates *codeia* in half- to one-grain doses.

III. DILATATION IN RENAL DISEASE

Chronic dilatation of the heart in renal disease may reach an extreme degree and be complicated by great dropsy.

There are *two important indications* in the treatment of myocardial affections due to disease of the kidneys. If the blood-pressure is continuously high and the habits of the patient not conducive to the sparing of the renal tissue, it is clear that the heart will be overtaxed, the hypertrophied muscle exhausted, and the terminal stage of dilatation accelerated. On the other hand, if the heart is feeble and the blood-pressure low, there will be a tendency for uraemia to supervene.

Accordingly, in the stage of hypertrophy the heart must be spared by the careful elimination of the retention products due to the renal disease.

As little meat as possible should be taken. This is a very different precaution to its prohibition, which experience has shown does not by any means always succeed well. The particular constitution of the patient must be studied. Some renal cases are confessedly better without any butcher's meat, others gain in health and strength by a moderate allowance.

The skin must be kept active by daily exercise. Turkish baths suit some cases well, and relieve the kidneys greatly. If the blood-pressure is persistently high a mercurial followed by a saline in the morning may be given, and a course of

nitro-glycerine or *sodium iodide* combined with a *bitter* taken for some weeks.

When the heart begins to show signs of dilatation, immediate rest and prompt treatment of the renal condition may arrest further breakdown.

A mixture containing *digitalis* and *nitro-glycerine* (10) is indicated with or without *strychnine*.

When there is severe dilatation, the condition is often complicated by such uraemic symptoms as asthma and vomiting, or by acute over-distension of the right side of the heart or dropsy, and each individual case will need special management. The uraemic symptoms will need to be combated in order that the *cardiac tonics* may take effect, and vomiting must be checked by *eliminants* and *gastric sedatives* aided in very severe cases by *rectal alimentation*. For the treatment of right heart failure, vide Chapter ix, p. 158. It frequently happens that improvement follows up to a certain point and then the condition remains stationary. This may mean that the heart has been strained to the utmost, and will no longer react to any remedies, in which case it is advisable to *stop all medicine* for a short time; or, on the other hand, dropsy may be the obstacle.

Fluid, especially if in the right pleural cavity, should be aspirated, and if the abdomen is distended with ascites, and the urine scanty, and the heart and lungs oppressed as a result, *paracentesis* is indicated.

The *dropsy* in these cases is cardiac, though doubtless aided by the disordered nutrition of the capillaries, the result of the renal disease, and this will be a convenient place to deal with the chief indications for treatment in cardiac dropsy, whether the result of chronic valvular disease, simple dilatation, or dilatation secondary to renal disease.

CARDIAC DROPSY

The fluids ingested should be restricted, and if there is deficient elimination of chlorides in the urine common salt should also be reduced in the diet.

Cardiac dropsy may be removed by producing *free diuresis*, or by *catharsis*, or by multiple punctures of the skin and *aspiration* of serous cavities. Free *diaphoresis* is also an aid, and in practice a combination of these methods is used.

Many of the cardiac tonics are diuretics, notably digitalis and squills.

The combined method is well illustrated by the use of the pill containing *mercury*, *digitalis*, and *squills*, the combination of squills and digitalis being particularly effectual (23). If the *infusion of digitalis*—the best preparation for the purpose—is used, it should be pushed after a preliminary opening of the bowels. If thus pushed and well borne, in *three days* some result should begin to be seen in an increase of the urine. If not, it may be that a pleural effusion is embarrassing the heart, and requires paracentesis.

Caffeine (19) may be injected under the skin, in other cases, or combined with digitalis.

Apocynum in two-minim doses of the fluid extract has succeeded where other remedies have failed; but Broadbent writes guardedly as to its safety.

Diuretin has been advocated in doses of fifteen grains every four hours.

Prescriptions 22 (a) and (b) may be of assistance if digitalis fails.

Pleural effusions should be withdrawn with special care, and stimulants always be in readiness. The engorged and damaged lung beneath may give rise to trouble, and paracentesis start a most harassing cough with the expectoration

of blood-stained oedema fluid. An injection of *morphia* and *atropine* should be given if this dangerous event arise.

Abdominal paracentesis. The fluid should be withdrawn slowly, with careful aseptic precautions and after emptying the bladder. A many-tailed binder is placed in position and gradually tightened as the abdomen is emptied.

The *connective tissues* are dealt with by multiple puncture, Southey's tubes, or incisions.

In *renal cases* the fewer wounds the better, for erysipelatous inflammation easily arises. When the oedema is more solid than usual, small punctures are useless. If the patient is restless Southey's tubes are not indicated, but they are very convenient, when effectual, on account of their cleanliness. Great attention must be paid to antiseptic details in these operations.

THE NAUHEIM OR SCHÖTT TREATMENT

The two essentials are the baths and the movements. Vide Appendix iii.

The treatment can be obtained in England or on the continent. It is most successful in dilatation of the heart of moderate severity, the result of infections, for example, influenza, or of imperfect nutrition, as in anaemia and debility. In valvular disease, the tone of the myocardium may be improved, and though in no way curative of the valvular lesion the exercises are useful preliminaries to more active movement.

The cold light of hospital observation does not perhaps discover such remarkable effects from the treatment as might be expected, but the altered scenes, the regimen, and the magic of the word 'cure', all no doubt take some part in the great improvement that has been frequently recorded

in suitable cases from this method of treatment. The reader must not be influenced by startling diagrams of heart outlines, but rely more upon the evidence that many sufferers from heart disease have received considerable benefit from a carefully elaborated combination of baths, exercises, diet, rest, and change of scene.

It will be clear then that severe aortic regurgitation, aneurysm and advanced fatty degeneration of the cardiac muscle are not likely to benefit; and the dangers of long journeys and strange surroundings, and the sadness of death in a foreign land should not be forgotten.

The baths at Nauheim are very rich in free carbonic acid gas and mineral matters. By their use the action of the heart is diminished in frequency, the impulse strengthened, and the area of cardiac dulness diminished. Nevertheless a diminished area of cardiac dulness after a saline bath is not necessarily an evidence of true improvement. It may only be a transient phenomenon, and then not entirely dependent upon a diminution in the size of the heart, but upon increased expansion of the lungs.

CHAPTER XIV

NEURO-MUSCULAR AFFECTIONS OF THE HEART

Sensory disturbances—Cardiac pain—Causation of—The tobacco heart—Tea-drinker's heart—The heart in gout—Cardiac pain from nervous causes and overstrain—Angina pectoris—Causation—Morbidity—Anatomy—Symptoms—Diagnosis—Prognosis—Treatment—Motor disturbances—Of rate—Infrequency of pulse—The Stokes-Adams syndrome—Symptoms—Prognosis—Treatment—Tachycardia—Causation—Symptoms—Prognosis—Treatment—Disturbances of rhythm—Disturbances of force—Palpitation—Intermission—Treatment of palpitation.

THIS is the most artificial and the most difficult chapter in heart disease, for it is concerned with conditions which may appear and disappear without any definite signs of an organic lesion, and with others that are certainly the result of permanent disease, but which in these particular instances manifest symptoms that point to disturbance of the neuro-muscular apparatus.

They may be divided into:—

1. Sensory disturbances—under which will be included the important disease, angina pectoris.
2. Motor disturbances: (a) of rate; (b) of rhythm; (c) of force.

Throughout this chapter, accepting obvious criticism, free use will be made of the clinical significance of the term disease.

I. SENSORY DISTURBANCES

A. Cardiac Pain

This may result from organic heart disease, or from causes which do not necessarily produced organic lesions.

Group I. The result of organic heart disease. Frequent allusions have been made in the preceding pages to the occurrence of pain in organic heart disease, and some of the more important examples may be summarized here:—

1. Affections of the pericardium, notably acute pericarditis.

2. Affections of the myocardium, whether fatty, fibroid, gummatous, or the result of growths.

3. Affections of the endocardium, notably aortic regurgitation and stenosis.

Group II. Toxic causes. These may produce organic disease, but this is not an invariable result.

(a) Microbial toxins, as in diphtheria and influenza.

(b) Chemical toxins, such as tobacco, alcohol, and gout.

Group III. Nervous causes.

Group IV. The result of cardiac overstrain.

Group V. The result of embarrassment, from gastric or general abdominal distension.

Cardiac pain is most commonly referred to *the apex of the heart* but may be felt over any part of the precordial area, and is often associated with a tenderness on pressure which may be exquisite. A frequent site of tenderness is immediately under the left nipple. In character the pain is dull or stabbing, or agonizing and terrible as in angina pectoris.

Group I and Group II (a) of the causes of cardiac pain have been already considered.

Group II (b). Causes due to chemical poisons.

The effect of tobacco on the heart. Sir Lauder Brunton attributes the action of tobacco not only to the nicotine, but to the pyridine and picoline bases. These are present in larger proportion where a pipe is used than in cigar-smoking.

The first effect of tobacco upon the circulation appears to be a rise of blood-pressure, which in chronic poisoning is followed by a lower blood-pressure than normal.

Intermission of the heart, palpitation and pain are the most frequent results, and sometimes there is a persistent irregularity in force and frequency. There is undoubtedly a great idiosyncrasy to tobacco-poisoning, and ill effects are most often met with in those who come from a non-smoking family. In these cases most alarming and sudden syncope may occur, or severe angina, especially if the smoke is inhaled. Such people can often smoke better after a full meal, and may be quite unable to do so while walking in the open air.

Treatment. There can be no doubt that those who are intolerant of tobacco should give it up. Many, however, will not do so, and these should be advised to smoke little, and never on an empty stomach, and they should use the mildest tobacco.

When the heart is irregular, all smoking should be interdicted, and the patient told that it may be some months before it will be safe to attempt its resumption. Symptoms of angina demand a complete prohibition of tobacco for many months, and if the patient can face this he will probably be persuaded to do what is even more wise, give up the habit altogether.

A prescription of *tincture of nux vomica*, *bromide of ammonium*, and *liquor arsenicalis*, as advised by Gibson, may be given for a month or six weeks.

Tea and coffee. The most usual effect upon the heart is acceleration by virtue of the caffeine contained in these substances, but any action is also complicated by the tendency of tea to cause atonic dyspepsia.

On this account the symptoms are usually complicated, but a prominent feature is *troublesome palpitation with an intense feeling of prostration*.

Tea and coffee are generally relinquished under orders with greater facility than tobacco or alcohol.

The heart in gout. This subject is notoriously vague and difficult, but there is sufficient evidence to enable a useful classification to be made of heart affections in gout:—

1. Cardiac affections, secondary to renal disease in gout. These have already been considered.

2. Cardiac affections due to degenerative lesions of the aorta, coronary vessels and myocardium, for which the reader is referred to Chapter xii.

3. Angina pectoris, described in the next section, may certainly be associated with the gouty state.

4. The vague group of cardiac affections which come into this particular section, are briefly—attacks of cardiac pain with palpitation associated with gastric disturbances. They are possibly examples of incipient angina pectoris, but may undoubtedly occur in patients who never develop the true disease. Irregularity of the pulse, increased rapidity with intermission, and fluttering of the heart, are other conditions which may occur, sometimes over long periods and sometimes paroxysmally. These may have no grave significance.

Treatment. The *functional gouty affections* must be distinguished from the organic lesions associated with chronic renal disease and angina pectoris. In many of these cases the

diet and digestion need attention, and if there is temporary high tension, *mercurials* and *salines* are indicated. *Alkalies* and *carminatives* combined with *colchicum*, given after meals, and later a course of *alkalies* (27), *arsenic* and *nuxvomica*, will improve the gouty state and general vascular tone. For the attacks a rapidly *diffusible stimulant* is indicated (24).

Group III. Nervous causes. Among the third or nervous group may be placed cases dependent upon *reflex disturbance*, the result of disease elsewhere, such as uterine affections or floating kidney. In other cases there are apparently disturbances of the vaso-motor mechanism with sudden alterations in the peripheral resistance. This in its most pronounced form constitutes Nothnagel's angina vasomotoria. Neurasthenic patients may among their multitude of symptoms suffer from cardiac pain, and other precordial sensations which can hardly be called painful. Lastly, sudden shocks, hysteria, sexual excess, great grief, and other violent emotions may be the exciting causes.

Group IV. Cardiac overstrain. In the fourth group the result of overstrain, although pain may be a marked feature, the disturbance of the action of the heart is of more importance. These conditions have been considered under myocardial disease.

Treatment. The treatment of cardiac pain must depend entirely upon its cause. In the first place it is of great importance not to alarm those patients who have no serious lesion by attaching over much importance to this symptom. In neurasthenic and hysterical cases much may be done by obtaining the confidence of the patients and allaying their fears. Disorders of the digestive system and pelvic organs must be appropriately treated. In the nervous cases *general tonics* are indicated, and local applications such as

mustard leaves, belladonna plasters, or even a blister are often useful.

B. Angina Pectoris

There are two ways in which angina pectoris may be treated of in writings upon the subject. It may be considered as a form of heart pain, a symptom of many different cardiac lesions, or it may be considered as a clinical disease, in which pain is the most striking feature. Convinced that in spite of some obvious difficulties the conception of angina pectoris as a clinical disease is the most instructive one, I shall adopt this plan. No distinctions will be drawn between false and true angina; these are both looked upon as examples of cardiac pain, and whether that pain is indicative of angina pectoris or not will depend upon the presence or absence of the other manifestations of that disease.

Ætiology. *Heredity* is of importance, and men are affected more frequently than women.

Angina pectoris seldom occurs before *middle age*, although it has been recorded in childhood. *Gout* and *syphilis* and infections such as *influenza* are additional predisposing causes.

Tobacco, coffee, and tea are probably also excitants in subjects who are predisposed. With regard to climate and season, it would appear that *cold* favours the occurrence of attacks. *Mental strain, sudden shocks, dyspepsia, and constipation* are also provocative.

The morbid anatomy. A number of different lesions have been recorded. The difficulty, however, is to decide upon the exact cause of the pain.

Huchard has done a good service in his classical work on heart disease by making the definite statement that angina pectoris is in reality a coronary angina, the result, that is

to say, of disease, organic or functional, of the coronary vessels. This, though open to doubt, brings into prominence a most important factor in the disease. Among the lesions that have been recorded in fatal cases are :—

I. Changes in the first part of the aorta, the result of

- (a) Direct injury.
- (b) Acute aortitis.
- (c) Atheroma.
- (d) Aneurysm of the intrapericardial portion of the aorta.

II. Changes in the coronary vessels, the result of

- (a) Narrowing of the openings from disease of the aorta.
- (b) Acute arteritis.
- (c) Endarteritis obliterans.
- (d) Atheroma.
- (e) Coronary embolism or thrombosis.

III. Changes in the valves.

- (a) Aortic stenosis.
- (b) Aortic regurgitation.

IV. Changes in the myocardium.

- (a) Fatty.
- (b) Fibroid.

V. Degenerative changes in the cardiac ganglia and cardiac plexus.

VI. In a few cases no lesion has been discovered.

It is evident from a review of the causes that many of the lesions will explain the fatal issue. The difficulty, as before remarked, lies in the explanation of the paroxysmal pain, for coronary disease, aortic lesions, and myocardial degeneration may all occur without this symptom. The pain has been variously ascribed to *cramp* of the heart and *neuralgia* of the cardiac nerves. Clifford Allbutt has called attention to its peculiar character and wide distribution, and has emphasized its distinctions from the usual forms

of cardiac pain. He localizes the pain in the *first part of the aorta*, a view to which I incline.

Huchard looks upon the pain as the result of ischaemia of the heart from a relatively deficient blood supply due to *disease or spasm* of the coronary vessels, and has supported this by giving instances of angina associated with intermittent claudication, a condition of *ischaemia* of the limbs, with cramps and temporary loss of power. As Sir W. Broadbent points out, there cannot be a general cramp or spasm of the heart, for the pulse would under these circumstances fail. It is possible that there may be an irregular and partial contraction of the muscle, but the point insisted on by Allbutt that dyspnoea is not a feature of severe angina pectoris, makes me doubtful whether the cause of the pain lies in the heart itself, and inclines me to his view that the pain starts from *the first part of the aorta*. Whether or not there need be actually visible aortic disease is doubtful, for it would be reasonable to believe that the expansion and contraction of the aorta, working harmoniously, as it does, with the contraction and diastole of the heart, may be disturbed by disease without any very gross structural lesion.

Symptomatology. There are clinically two grades of angina pectoris—the severe and the mild.

A. The severe. The first symptom is usually *pain on exertion*, or after fatigue.

This pain is severe, often agonizing, and usually described as rending, crushing, or gripping in character. Its principal seat is over the upper part of the sternum, whence it often radiates to the back, and down the left arm, often also upward to the back of the neck and head, and occasionally down the right arm. The face is *pale, livid, sweating, and anxious*; a sensation of *impending death* is generally recognized as one of the most remarkable and characteristic symptoms

of the disease. In these severe attacks the rule, with, however, exceptions, is for the patient to *stand still* leaning over some support and holding his breath. The *mind* is generally *clear*, unless there should be *syncope*. If, however, the suffering is very intense, and has been repeated, there may be transient delirium, and if, worn out by suffering, the patient is sinking, there may also be some wandering of the mind.

The physical signs. The *pulse* varies during the attack, sometimes it is but little altered, sometimes there is vasoconstriction. Sometimes, on the other hand, it is soft and feeble throughout. Usually quickened, all the heart-beats may not reach the pulse, which then is apparently slowed.

The *heart* is difficult to examine under such alarming circumstances. The action may or may not be accelerated, and in some instances an apical systolic murmur has been detected.

The *duration* of the attack may be a few minutes, or in recurring waves it may last for some hours. A remarkable feature is an *eructation of wind* often with loud belching which frequently marks the close of an attack, and reminds one forcibly of the same occurrence in asthma. Similarly there may be, at the end of the paroxysm, the passage of a *copious amount of pale urine*.

There is profound shock resulting from such suffering, and, although there may be a wonderful power of rallying, on the other hand there may be in some cases no true recovery, and the patient pass away without further suffering, or die suddenly from a slight recrudescence.

B. The milder form of angina pectoris. This is more closely associated with *vasomotor spasm*. There can be no sharp distinction drawn between the two forms, because if the spasm occurs where the heart is diseased, from myocardial change or valvular lesions, this spasm may determine

a severe attack. Nevertheless, it is recognized that such spasm may occur when the heart is strong and even young, and that the recovery from this angina may be rapid and complete. In some of these cases the circulation has often been for many years defective, although the blood-pressure is frequently high. *Digestive disturbances* are also frequent. Both males and females suffer, but there is a marked increase in the number of females at the time of the menopause. The attack is sudden: there are *substernal pain* and a sense of oppression. Occasionally also there is *syncope*. *Air hunger* is a prominent symptom, and there is generally *restlessness* and active movement during the attack. The paroxysms may occur at first frequently, but under treatment usually subside gradually.

As remarked, between these two types there are intermediate grades, and it is essential to detect as far as possible the extent to which the paroxysms are the result of nervous causes that are preventable.

Again, in some of the dangerous cases the attacks alter in character. The pain becomes less severe, and the signs of cardiac failure more prominent. In such cases a fatal syncope is to be expected. Between severe attacks also there may occur abortive ones, some of which may be the result of early precautions taken to arrest their full development.

Diagnosis. The clinical disease angina pectoris is characterized by its tendency to occur at and after middle life, and in men rather than women; by its remarkable and severe pain, which is associated with and may be replaced by syncope, and which is often first noticed after over-exertion; and by the occurrence of the sensation of impending death. It is a condition essentially paroxysmal, and liable to recur with increasing frequency after the first

attack. Lastly, disease of the first part of the aorta and coronary vessels is often found in fatal cases.

The diagnosis when the disease has these features is an easy one, but some of them are often lacking, and then there may be considerable doubt. Among the most likely conditions to be confused are :—

1. Cervico-brachial neuritis or neuralgia.
2. Intercostal neuralgia.
3. Rheumatic or gouty intercostal neuritis.
4. Renal and hepatic colic.
5. Spasm of the pylorus.
6. Aortic aneurysm.

1. In *cervico-brachial neuritis* there are usually tender points where the various nerves pierce the fascia. There may be herpes, glossy skin, and well-marked muscular atrophy. The pain is greatly increased by movement of the limb.

2. *Intercostal neuralgia* and (3) neuritis resemble pleurisy more closely than angina pectoris; there is not the sense of impending death, or the profound cardiac disturbance.

4. In *renal colic* the distribution of the pain is different, and there are as a rule no cardiac symptoms other than syncope from the pain; the same applies to hepatic colic.

5. *Spasm of the pylorus* gives rise to paroxysmal epigastric pain, which may pass through to the back; this is accompanied by gastric dilatation and flatulence. Such a condition may give rise to angina pectoris in those who are sufferers from the disease, but the distribution of the pain in the condition itself differs from that of angina; the symptoms are digestive rather than cardiac, and the apprehension of death is not present.

6. *Angina pectoris* may be associated with aneurysm of the first part of the aorta, and the aneurysm may be entirely overlooked. On the other hand an aneurysm may cause

pain from pressure, and this pain be mistaken for angina pectoris. Search must be made for a pulsating tumour, and an X-ray photograph of the chest taken in difficult cases.

The sex and age of the patient, the relation of the first attack to exertion, and the attitude and aspect of the patient at the time of the paroxysm should all be taken into account when deciding upon the diagnosis of angina pectoris. Above all the dangerous mistake must be avoided of under-rating the gravity of the symptoms because, as often occurs, there is a clear element of neurosis in the case.

Prognosis. This must necessarily be uncertain, for it is frequently associated with myocardial affections that can only be guessed at rather than diagnosed.

Much importance must be attached to any definite excitants of the attacks that are preventible, be they digestive, mental, emotional, or physical. Their correction may entirely alter the outlook.

If the blood pressure is high and there is marked vasoconstriction the outlook is better, for these can be suitably treated. When the attacks are severe, with no obvious explanation for their occurrence, and the heart is feeble, the prognosis is most serious. And this is the more true if the syncopal element is prominent.

Treatment. The first step is to relieve the paroxysm.

The remedies that come first are the nitrites.

Nitrite of amyl in 5-minim doses in capsule is the most rapid in its action.

Nitro-glycerine is slower in action, but more prolonged in its effects. This is conveniently dispensed in tablet form, each containing 1 minim of a 1 per cent. solution.

Erythrol tetranitrate is even more prolonged in its effect. This can be dispensed in tablet form, each one containing 1 grain. Dose 1-8.

Theoretical considerations would point to these remedies acting most successfully when there is arterial spasm, but they are also effectual when there is no marked spasm.

These remedies *excite the action* of the heart in addition to relaxing the peripheral arterioles, and this effect is sometimes a bar to the employment of erythrol tetranitrate. The particular preparation in use should be carried by the patient when the attacks are severe in a convenient form for instant use. Milder attacks may be averted by a diffusible stimulant (24).

When the *blood-pressure is low*, the heart feeble, and the stomach distended with flatulence, these drugs may disappoint, and better results are gained by the use of *strychnine*, *carminatives*, and *diffusible stimulants*, followed by an hypodermic injection of *morphia* and *atropine* to ease the pain. *Oxygen* should be administered, but the mouth-piece not directly applied, and the gas should, if possible, be warmed.

There is always a risk in giving *morphia* to these cases, on account of the initial period of shock after the injections and before the drug takes effect. At first one-fourth of a grain can be given, but if the attack is prolonged the dose must be raised and the risk taken. Balfour preferred the use of *chloroform* by inhalation.

In some cases which are not very severe a mustard leaf or turpentine stupe applied to the precordium may give relief.

When the attack is over, strict injunctions must be given that the patient should maintain absolute rest for at least three days, for, however quick the rally may be, such a shock to the heart leaves its mark. Three days has been given as a time limit, but, in many cases, this is only a mere safeguard for the doctor, in order that he may get a good grasp of the nature of the case. He will,

after that time has elapsed, judge from the pulse, symptoms, and physical examination of the heart, how soon to allow his patient to resume his occupation. In severe cases, the result of organic disease complicated with mental overstrain, a long rest may bring about a most satisfactory result.

The general management of a patient subject to attacks of angina is of great importance.

The habits of life must first be taken into account.

The gouty man of full habit must be instructed to take regular exercise, and a diet prescribed (vide Appendix). The neurasthenic will need rest, and cases of this kind, if they follow influenza, tobacco poisoning, or other toxic causes, will benefit from rest combined with massage, and later graduated exercise and saline baths (vide Appendix).

Sudden chills and coldness of the extremities are particularly liable to induce an attack; cold sheets at night, or a cold bath in the morning are sometimes responsible, and the patient should be duly warned against them.

In the same way the diet must be adapted to the nature of the case. When needful the appetite must be restricted. On the other hand, there are cases in which the meals are hurried and insufficient. These must be made regular and wholesome, and the patient must rest before, and for a short time after them.

In some cases, and these are the most frequent, eliminant drugs are required.

If the blood-pressure is high, a dose of *blue pill*, followed by a tumbler of *Vichy water* in the morning, is indicated once in three weeks, or oftener if necessary. Should gout be an evident factor, *colchicum* combined with *alkalies* is employed for the robust, and if more *tonic* remedies are required a gentian or cinchona mixture, given with a 5-grain dose of potassium iodide, may be ordered.

In angina of the less severe type there is frequently much mental disquietude, and then the *bromides* are of value.

In every case of angina pectoris the *digestion* will require careful management. Flatulence and dyspepsia with constipation are treated on the usual lines, and although, as indicated above, an occasional mercurial is often useful, *simple aperients*, only, are needed for routine administration. The cases in which the heart is feeble and the blood-pressure low may benefit much from *strychnine*, and if there is pain, from a course of *arsenic* (25) combined with a bitter. Sir W. Broadbent praises *phosphorus* in the angina associated with aortic regurgitation. *Caffeine* is indicated when the urinary secretion is diminished.

Digitalis and *strophanthus* are generally thought to be somewhat hazardous in the degenerative cases with a fatty heart and increased blood-pressure, but in some of the toxic cases in younger people they may be very useful.

A firm stand must be taken against an *abuse* of such remedies as amyl nitrite and nitro-glycerine. If they are employed by the patients to enable them to take liberties with their physical and mental strength, there is a danger of their losing all effect and of precipitating a fatal syncope.

II. MOTOR DISTURBANCES

(a) Disturbances of Rate

Under this section will be described two clinical conditions, one characterized by remarkable infrequency of the pulse, the Stokes-Adams syndrome or paroxysmal bradycardia, the other characterized by great frequency of the pulse, and termed tachycardia or paroxysmal tachycardia.

A. Infrequency of the pulse. This is sometimes called bradycardia, but an infrequent pulse is only one symptom

of many different affections, and for this reason bradycardia will be used here for the paroxysmal type alone.

An infrequent pulse may be :—

1. Physiological. Some individuals have habitually a pulse rate under 60, and may have inherited the peculiarity. Possibly such a condition is more liable than usual to be associated with degenerative cardiac lesions, but it is certainly compatible with a long and useful life.

2. It may result from various nervous affections, as for example, cerebral compression, neurasthenia, and melancholia.

3. Or from reflex causes, such as the severe pain of colic.

4. Or from infective diseases, such as, for example, influenza or diphtheria.

5. From organic heart disease, e.g. aortic stenosis.

6. The most important and characteristic cases of infrequent pulse are those which result from disease of the heart coupled with arterio-sclerosis, and it is in this group that most cases of the Stokes-Adams syndrome occur.

THE STOKES-ADAMS SYNDROME, OR PAROXYSMAL BRADYCARDIA

These names are given to a clinical disease characterized by infrequent pulse, associated with vertiginous, syncopal, and respiratory attacks, or in some cases with epileptiform or apoplectiform seizures.

According to Osler, not all cases are of the cardio-vascular type. A few are *post febrile*, occurring after typhoid, influenza, or rheumatism, and a few are due to organic or functional nervous disease.

The condition is met with most frequently in males over fifty years of age.

The pathology of bradycardia is difficult. Krehl explains the infrequency of the pulse as the result of sclerosis of the coronary vessels, but Huchard's view that it results from sclerosis of the cerebral arteries is more widely accepted. Tupier believes that the epileptiform seizures are the active cause of the cardiac infrequency, but there appears to be no doubt from clinical observation that the slowing of the heart precedes the epileptiform attacks.

In a certain number of cases of the Stokes-Adams syndrome, disease has been found in the auriculo-ventricular bundle of His, and the immediate cause of the infrequent pulse has been attributed to imperfect conduction of the auricular impulses to the ventricles, that is to a condition of partial or in some cases even complete 'heart-block'; vide Chapter i, p. 14.

Symptomatology. The symptoms are grouped by Osler under these headings:—

1. The cardio-vascular.
2. The nervous.
3. The pulmonary.

1. The cardio-vascular. The pulse is in some of these cases always infrequent, but at the time of the paroxysm the rate may fall as low as twenty or even fewer beats per minute. This constitutes *true bradycardia*.

In others it is slow, but auscultation of the heart detects a number of *rapid inefficient ventricular systoles*. This is a false bradycardia.

Thirdly there may be *cardiac arrest*, a most remarkable and alarming phenomena. In one of Osler's cases no cardiac movement was detected for thirty-five seconds. This condition is sometimes called 'heart-block'.

Vaso-motor symptoms such as sweating, numbness, and tingling occur in a certain number of these cases.

2. The nervous symptoms. The most prominent are : vertigo, syncopal, apoplectiform and epileptiform seizures. These have already been described in the section on fatty heart, p. 203.

3. The respiratory symptoms include cardiac asthma, oedema of the lungs, and Cheyne-Stokes respiration.

Prognosis. This is grave ; some of the post-febrile cases recover, but the majority, associated as they are with degenerative lesions, do not tend to improve.

It is not possible to foretell the duration of life with any precision, but some of these cases have lived for six years, or even longer, after the first definite symptoms. The end is usually sudden.

Treatment. In the post-febrile cases prolonged rest is important, and in all cases a quiet life is very essential. When there is arterio-sclerosis a course of *sodium* or *potassium iodide* (26) in moderate doses is indicated, and if the tension is high, *nitro-glycerine* and *amyl nitrite* are serviceable.

For the syncopal attacks *brandy*, *diffusible stimulants* (24), and *oxygen* are necessary, and in one case under my care, in which the attacks usually occurred in the morning shortly after breakfast, great advantage was gained from a full dose of *strychnine* taken immediately after the meal. The patient did not attempt to dress until the action of the drug had taken effect. *Atropine* has been suggested to combat the tachycardia, but at present its value is not established, and it is a drug that is often unpleasant and difficult to take.

B. Excessive frequency of the pulse and paroxysmal tachycardia. Increased frequency of the pulse may be produced by various causes which are summarized below.

1. Physiological. In some individuals the pulse may habitually beat at 80, 90, or 100 to the minute.

2. High fever and acute infections. Notably scarlet fever.

3. Organic heart disease, particularly mitral stenosis among the chronic valvular lesions, and all acute inflammatory affections.

4. Bacterial and other toxins, such as those of diphtheria, scarlet fever, and rheumatism.

5. Drugs, and active principles, such as strychnine, atropine, tobacco, and alcohol.

6. Nervous causes. Functional as a result of emotion or neurasthenia. Organic, as in some cases of spinal disease.

7. Cardiac overstrain.

8. Graves' disease.

9. Paroxysmal tachycardia.

PAROXYSMAL TACHYCARDIA OR TACHYCARDIA (ALLBUTT)

This will be made the landmark in this section, as was the Stokes-Adams syndrome in the preceding one.

This is a condition in which there is a sudden and remarkable increase in the frequency of the pulse and heart, which may or may not be the seat of organic disease. There is not the tumult of palpitation or the irregularity in force and volume.

It is usually associated with the nervous temperament. Both sexes are affected, and it may commence in childhood or in adult life. The pathogeny is obscure. It would appear as most probable that there is some derangement of the inhibitory mechanism. In a case of Mackenzie's changes were found in the auriculo-ventricular bundle. In a considerable number changes of various kinds have been found in the myocardium.

The onset may date from a sudden over-exertion or emotion, but no such cause may be forthcoming.

Symptomatology. The tachycardia starts abruptly, usually ends abruptly, and seldom lasts longer than three or

four days. Among the symptoms are *lassitude*, *exhaustion*, and *restlessness*. There is a sense of oppression and tightness in the chest. Allbutt records a case of *aphasia* at the height of the paroxysm. With the abrupt cessation there is a sensation of relief, and a period of exhaustion followed by recovery. In fatal cases death occurs from syncope.

The physical signs. During the attacks the *pulse* frequently cannot be counted, and the number must be taken by auscultation over the heart. In Bristowe's case, 808 heart-beats per minute were recorded.

The *cardiac sounds* are tic-tac, and there may or may not be a systolic, whiffing, mitral murmur. When the condition is continually relapsing or the attack unusually severe, signs of dilatation may appear, and later still dropsy, albuminuria, and oedema of the lungs. The duration of an attack varies from a few minutes to four or five days, or according to some writers even longer.

Prognosis. Herringham points out that if tachycardia persists after the age of thirty years, the patient will probably not live to be over the age of fifty; there are undoubtedly exceptions, and with increasing years the condition tends to wear itself out.

Treatment of frequent pulse. *Rest* and mental quiet are necessary, and in cases of pendulous abdomen and floating kidney, an abdominal belt is of value. *Respiratory exercises* are also indicated in cases of moderate severity.

Gastric derangements should be corrected, and in mild cases this, with *carminatives* (25), will suffice. *Digitalis*, *ammonium bromide*, and *nepenthe* may be presented in 4-hourly doses in the more severe forms. Others prefer *belladonna*; this may be combined with *bromide* and *iodide of potassium* (26), and the *belladonna* pushed to the limit of tolerance. Barr has obtained success with *nitro-glycerine*

and nitrite of amyl. In some cases treatment does not appear to influence the condition. On the other hand, there are others in which an attack has been arrested by the patient taking a deep inspiration, fixing the chest, pressing the arms against the sides, and then making a forced expiratory effort.

(b) Disturbances of Rhythm

The conditions in which this occurs have been considered incidentally in the foregoing pages. Gibson classifies the causes under two groups :—

1. Intrinsic or cardiac causes.
2. Extrinsic or nervous.

Among the cardiac causes are :—

1. Toxic as in pneumonia, influenza, &c.
2. Due to such poisons as alcohol, tobacco, and tea.
3. Overstrain.
4. Organic heart disease. Acute and chronic affections, peri-, endo-, or myo-cardiac.
5. Prolonged high peripheral resistance.

The nervous causes arise

- (a) From the disturbances in other viscera ;
- (b) From direct nervous influences, functional in nature.

(c) Disturbances in Force

These are illustrated by palpitation and tremor cordis. Syncope has already been mentioned.

Palpitation. This may occur in organic heart disease, particularly in advanced mitral stenosis, but is most characteristically met with apart from organic disease.

It is more frequent in the female sex, and is often reflex in origin. Disturbed digestion, mental overstrain, over-

lactation, anaemia, and uterine disorders are among the common exciting causes.

SYMPTOMATOLOGY

The attacks may come on by day or night, and are often sudden in onset. The face flushes, and there is a distressing sense of turbulence in the chest which leads the patient to press the hand over the precordial area. The *pulse* is exceedingly rapid and irregular in force and frequency. The *cardiac excitement* makes auscultation unsatisfactory. A systolic mitral murmur may appear, and disappear again with recovery.

At the end of the attack a quantity of pale urine may be passed.

Intermittence of the pulse and fluttering heart. In some cases intermission of the pulse is unrecognized by the patient; in others there is a horrible flutter in the chest as if the heart had turned a somersault. This sensation occurs synchronously with the dropped beats.

If intermission occurs in degenerative myocardial affections or in advanced aortic regurgitation, the sign is one of importance. In cerebral diseases, or in acute diseases in the elderly not primarily cardiac, intermission is also a warning of serious meaning. When it results from hurried meals, tea, smoking, and sedentary habits it is not serious, but is a sign of sufficient importance to demand prompt treatment. Gouty people are sometimes much troubled by fluttering heart, but the symptom then is more distressing than dangerous.

The treatment of palpitation. The cause must be determined.

If gastric, *bismuth* and *soda*, with a nightly cachet of

compound rhubarb powder are useful, and Gibson strongly recommends the combination of *strychnine* with *hydrobromic acid*.

Overstrain will be met by *rest*, *digitalis*, and *strophanthus*.

If a nervous breakdown is the cause, a prescription of *arsenic and iron*, combined sometimes with a complete rest cure, is indicated.

Tea, alcohol, and coffee should be forbidden.

CHAPTER XV

CONGENITAL HEART DISEASE

Development of the heart—The foetal circulation and the changes at birth—Pathology—Types of congenital heart disease—Pulmonary stenosis with patent interventricular septum—Symptoms—Diagnosis—Prognosis—Treatment.

IN this chapter no attempt will be made to describe all the numerous and sometimes extraordinary malformations that have been recorded, for many of these are of no practical importance to the physician. Attention will be mainly directed to those which are compatible with a life that may be of considerable duration.

A brief account of the development of the heart and the foetal circulation will assist in the easier comprehension of these forms of heart disease.

I. THE DEVELOPMENT OF THE HEART

The first sign of the formation of the heart is the fusion of the median parts of the two primitive anterior ventral aortae. This takes place in the third week, and the single tube thus formed constitutes the primitive heart. Upon this there appear four dilatations termed (1) the sinus venosus, (2) the auricle, (3) the ventricle, and (4) the bulbus arteriosus.

The *sinus venosus* ultimately forms a part of the right auricle and the coronary sinus, but at first it is separated from the auricle by a valve consisting of a right and left

segment. The right segment of this valve will form part of the Eustachian valve, the left one is merged in the inter-auricular septum.

The next event is an alteration in the shape of the primitive heart, by the occurrence of a *lateral V-shaped curve*, and in addition, an auriculo-ventricular curve which is antero-posterior in direction.

The result of these curves is to bring the ventricular part of the tube forward and to the right, and the auricular backward and to the left. From this curved single tube, *the four chambers* are formed by the development of septa.

The development of these septa is most easily understood after considering the changes in the auriculo-ventricular region. The passage between the single auricle and ventricle elongates into an *auriculo-ventricular canal*, and is then divided by the projection of two cushions, one springing from its anterior and one from its posterior wall. These, the *endocardial cushions*, fuse, thus making a right and left auriculo-ventricular opening. Meantime, the inter-ventricular septum springing from the lower limit of the ventricle grows up toward these cushions, and toward the septum that is forming in the bulbus arteriosus, which eventually divides the bulbus into the aorta and pulmonary artery.

The three septa, viz. the fused endocardial cushions, the inter-ventricular septum, and the arterial septum meet to form the *pars membranacea*, termed often in English writings, the undefended space. This spot in the fully-developed heart lies at the extreme upper end of the inter-ventricular septum immediately below the level of the auriculo-ventricular groove, and is in congenital heart disease sometimes imperfect, and then allows a direct communication between the two ventricular cavities.

The *inter-auricular septum* is more complicated in develop-

ment. There first appears from the back of the primitive auricle a septum, the *septum primum*, which descends toward the endocardial cushion, but separates from the roof of the auricle, leaving an open space above its upper border. There next appears, to the right of this, another septum, the *septum secundum*, springing from the roof and posterior part of the auricle, and extending downward to the centre of the cavity. Between these two septa is a space, the *foramen ovale*.

The development of the four-chambered heart is clearly a process of some complexity, and although it may be difficult to assign the cause for the production of malformations, there are many possibilities that may arise should this process of development be delayed or perverted.

II. THE FOETAL CIRCULATION AND CHANGES AT BIRTH

(a) **The Foetal Circulation.** In foetal life the blood is arterialized in the placenta and enters the foetus through the umbilical vein. This vessel on reaching the liver divides into two main branches. The larger enters the transverse fissure, breaks up in the liver, and reaches the inferior vena cava by the hepatic veins. The smaller passes directly into the inferior vena cava through the liver in a continuation of the umbilical vein, now termed the ductus venosus.

The blood in the inferior vena cava which is, in the main, arterial, meets in the right auricle the descending stream of venous blood from the head, neck, and upper extremities, but is deflected by the Eustachian valve into the left auricle and thence passes to the left ventricle, and is driven up to the head and upper extremities. The venous stream from

the head, neck, and upper extremities on the other hand passes directly into the right ventricle and is thence driven to the pulmonary artery. The greater quantity passes along the ductus arteriosus into the aorta beyond the point of origin of the left subclavian artery, and thence passes by the umbilical vessels to the placenta, and but little reaches the undeveloped lungs.

(b) **Changes at birth.** With the establishment of respiration there is a great increase in the pulmonary circulation, and simultaneously the umbilical vessels are tied in the cord. The ductus venosus and umbilical veins are filled with clots and disappear rapidly. The ductus arteriosus also atrophies, and is usually closed within the first fortnight of life. The foramen ovale, already at the end of foetal life much reduced in size, is now closed by the completion of the inter-auricular septum.

THE PATHOLOGY OF CONGENITAL HEART DISEASE

The causation is often obscure, and although it affords no explanation to describe a group as the result of malformation, this division will be adopted here, as convenient. It also serves to emphasize this gap in our knowledge of the aetiology, and to form a contrast to a group which is undoubtedly the result of intra-uterine inflammation.

There are accordingly two main divisions, which to some extent overlap one another.

Group No. 1. The result of malformation.

Group No. 2. The result of intra-uterine endocarditis.

Group No. 1. The Result of Malformation

This may be conveniently subdivided into:—

1. Cases in which the arrest of development has occurred in early foetal life.

In such cases the heart may consist of two chambers, an auricle and a ventricle, or possibly of two auricles and a ventricle.

These conditions are usually incompatible with life. Dixon Mann has recorded a case in which a man lived to be thirty-five years old with a heart consisting of one ventricle and two auricles, and one wonders as to the fate of the auriculo-ventricular bundle of His under such circumstances as these.

2. Cases in which the arrest has taken place at a later period. All four chambers are present, but are imperfect, and the large arteries may either be partially developed or misplaced.

3. Cases in which the defect occurs at the end of foetal life. As a result there may be premature closure of the foramen ovale or premature obliteration of the ductus arteriosus.

It is with the lesions of the later period of foetal life that it becomes difficult to decide the question whether the imperfections in structure are due to disease or malformation.

Group No. 2. The Result of Intra-uterine Inflammation

The most striking examples of this group are those in which the mother has, *during her pregnancy, suffered from rheumatic fever*, and the foetal valves are attacked as a result of direct infection. I am of opinion that the 'diplococcus rheumaticus' circulating in the maternal blood-stream is carried to the placenta and thence into the foetal circulation. It is conceivable that any bacterium which has the power of attacking the cardiac valves may be a cause of foetal endocarditis, but our most reliable facts are those in

connexion with rheumatism. Thus it is on record that in cases of rheumatic fever complicating pregnancy, a cardiac murmur has been detected in utero, and the infant has been born with heart disease. Vegetations have also been found upon the cardiac valves which were indistinguishable from those of acquired heart disease, and lastly I have found micrococci indistinguishable morphologically from the diplococcus present in rheumatism, in the mitral valve of a case that died within forty-eight hours of birth: the mother suffering in late pregnancy from rheumatic fever: a point of much interest.

The *valves on the right side of the heart* are especially liable to damage in this group, but this rule is not an invariable one, for the mitral and aortic valves may both be attacked.

There are many cases of congenital heart in which no more obvious cause is to be found than a history of ill-health, and possibly of syphilis, in the parents. Maternal impressions are also sometimes credited as a factor by the mothers of these children.

TYPES OF CONGENITAL HEART DISEASE

1. The most important congenital lesion is a complex one consisting of:—

1. A stenosis of the pulmonary artery from thickening or adhesion of the pulmonary valves.
2. A small opening in the undefended space between the two ventricles.
3. A patent foramen ovale.

The pulmonary stenosis may be a result of intra-uterine inflammation or of malformation.

Excluding the gross forms of malformation, other less common types are:—

PLATE XV



Congenital heart disease. The left ventricle is open, and a glass rod (B) is passed through the patent inter-ventricular septum. To the left and above is visible the minute opening (A) of the stenotic pulmonary artery.

*From a specimen in the Museum of University
College Hospital.*

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2. A patent foramen ovale alone. This frequently gives rise to no symptoms at all.
3. Pulmonary stenosis alone.
4. A patent inter-ventricular septum alone.
5. A patent ductus arteriosus.
6. Transposition or alteration in the arrangement of the main arterial trunks.
7. Lesions of the mitral, aortic or tricuspid valves.

The Symptoms of Congenital Heart Disease

These are as a rule well marked, and the most important are included in the subjoined table:—

1. Cyanosis.
2. Clubbing of the fingers and toes.
3. Laboured respiration, and paroxysms of disordered breathing.
4. Syncopal and epileptiform attacks.
5. Wasting or general retardation of growth.

Together with these there are the physical signs of organic heart disease.

1. Cyanosis is the most frequent and striking of all the symptoms, and for this reason congenital heart disease has been called 'Morbus Coeruleus'. The explanation of the cyanosis was thought by some to be dependent upon the mixture of arterial and venous blood, but there is conclusive evidence against its acceptance. Mann's case for example with one ventricle did not show this symptom. Neither can the view that it is the result of venous congestion be accepted. A more probable suggestion is Peacock's, that one factor at least is the small relative amount of blood aerated in the lungs owing to the pulmonary stenosis. This can only, however, be a factor. Another point to which attention was particularly directed by Toeniessen is a great increase in the number of

red blood corpuscles. Not only may this occur, but the specific gravity of the blood is also raised and the percentage of haemoglobin excessive. There result a concentration of the blood and increased viscosity, together with increased resistance of the flow of blood in the vessels, the result of the great number of red corpuscles. In consequence there is according to some authorities deficient oxygenation. Cyanosis has also been ascribed to a permanent dilatation of the superficial vessels.

Although cyanosis is a most important symptom of congenital morbus cordis, there are a considerable number of cases in infancy *which show either no cyanosis or so little that it may be overlooked.*

2. Clubbing of the extremities is a later symptom, and is generally preceded by redness and a shiny appearance of the skin of the terminal phalanges. When clubbing is marked there is usually much cyanosis. The soft tissues are implicated rather than the bones.

3. **Respiratory disturbances.** Dyspnoea on exertion is the most frequent, but there may also be paroxysms of disordered breathing, which are of great significance and are often associated with paroxysms of cardiac failure.

4. **Syncopal attacks** should always attract attention. They may vary in severity from a faint brought on by some definite cause, to the most alarming and prolonged attacks of unconsciousness in which the heart acts with great rapidity and the patient lies livid and at death's door.

5. **Epileptiform seizures** may complicate this condition. It is not unusual for the turbulent action of the heart to be the first symptom to attract the mother's attention.

During these periods of acute cardiac failure, there may be very great distress, and angina pectoris has been recorded even in children.

6. Particular emphasis must be laid upon the fact that in some infants **wasting** and refusal of food may be the prominent symptoms and should pallor rather than cyanosis be present, the nature of the case may be easily overlooked.

Older children and adults are generally *stunted* in growth.

The following are the physical signs of the most usual form of congenital morbus cordis.

1. **Enlargement of the heart.** This enlargement is not necessarily considerable, but should it be, it is the right side of the heart that is particularly affected. The enlargement of the deep cardiac dulness is, as a result, more circular in outline.

2. A *systolic thrill* which is felt most distinctly over the region of the pulmonary conus.

3. A *systolic murmur* with its maximum intensity in the third, left, intercostal space. This murmur varies much in intensity in different cases. It may be exceedingly loud and harsh and audible over the whole chest, front and back. On the other hand, in small and feeble infants it is sometimes most difficult to detect, partly on account of the restlessness and crying of the patient, and partly because it has a curious puffing character, which may easily be confused with the breath sounds.

The *pulse* is generally increased in rate but shows no special characters.

Bruits in the less usual types of Congenital Heart Disease

There are some other murmurs which need a brief mention.

(a) Should the *aortic, mitral or tricuspid valves* be damaged, there may be murmurs pointing to regurgitation or stenosis as the case may be, but it must be confessed that malformations of the most extraordinary nature may also give rise

to bruits which cannot be distinguished from these classical ones.

(b) A loud, shrill, systolic murmur, unaccompanied by a thrill, and with its maximum over the sternum at the level of the fourth costal cartilage, may occur in some cases of *patent inter-ventricular septum without pulmonary stenosis*. This condition has, however, also been associated by some observers with the murmur next to be described.

(c) When the *ductus arteriosus is patent*, there may be a continuous murmur occupying the entire cardiac cycle. It is both systolic and diastolic. The point of maximum intensity is in the third intercostal space on the left side close to the margin of the sternum. The character is remarkable. Starting piano in systole, by a rapid crescendo it reaches its maximum at the time of the closure of the pulmonary valves, and then dies away in diastole.

The Broadbents, however, in their manual describe a fatal case in which the murmur was only systolic, and consider the murmur described above as diagnostic of a patent inter-ventricular septum. It is very probable that these bruits differ in character in different cases.

Diagnosis. As a rule the diagnosis is easy. There is a danger, as has been already mentioned, of *overlooking the condition* in pale and wasting infants.

There may be some doubt also in older cases as to whether the condition is congenital or acquired. In such, stress must be laid on the history, the position of the murmur, and the symptoms. Pulmonary murmurs, if not functional, always suggest congenital lesions. *Acquired heart disease*, mitral stenosis in particular, may be associated with clubbing of the fingers, and acquired heart disease may also complicate the congenital lesion, and then the condition is most puzzling. The practical value of a distinction in these cases is not,

however, really great, for the problems of prognosis and treatment are very similar. It should be borne in mind, that acquired heart disease may occur at a very early age.

Functional basal murmurs in young children and anaemic infants cannot sometimes be distinguished from those due to congenital heart disease, until after death. In *rachitic* children also the diagnosis is sometimes not easy, but the rule should be made not to diagnose congenital heart disease in such cases unless there is good evidence of cardiac symptoms. Even cyanosis must be accepted with caution if the rickets is complicated, as is so often the case with bronchitis. It is perhaps not sufficiently recognized that a late systolic murmur with its maximum in the second left intercostal space is not a rare occurrence in children under five years of age. The heart in such cases usually shows no increase of size.

In *Cretinism* there may be extreme cyanosis of the extremities. The harsh skin, scanty hair, large tongue, spade-like hands, fat pads, heavy aspect and general backwardness, betray the condition, and thyroid extract cures the cyanosis.

The *Mongolian type* of imbecile may, on the other hand, be also the victim of congenital heart disease.

Lastly, in some cases of *retraction of the upper lobe of the left lung*, the result either of pulmonary disease, or of deformity of the chest, loud cardio-pulmonary murmurs may be confused with congenital murmurs. The entire case must be carefully reviewed before arriving at a conclusion and usually the distinction can then be drawn.

The Prognosis. Before summarizing the chief points in prognosis, some allusion must be made to certain important complications.

(1) *Sudden death.* In infancy, there may suddenly supervene a livid pallor, general surface coldness, and rapid death.

(2) *Cerebral thrombosis* may also occur in weakly infants, and doubtless it is encouraged by the feeble circulation. It may prove rapidly fatal.

(3) The supervention of *acute malignant endocarditis* is a danger which I have found more to be feared than tuberculosis in these patients when under twelve years of age. It is in some cases a rheumatic manifestation, for there can be no doubt that children with congenital heart disease are particularly liable to acute rheumatism; and often give a history to that effect.

The onset of this condition is attended by high fever, rapid and excited cardiac action, and sometimes by a definite change in the character of the murmur. Of even greater importance, when it is detected, is the appearance of murmurs differing in character from the already pre-existing congenital bruit.

(4) *Tuberculosis* is a serious danger, though its occurrence does not necessarily mean that the condition is incurable. Unfortunately, in most cases the tendency is to progressive spread of the pulmonary lesion.

(5) *Acute respiratory disorders* other than tubercular are also a great danger, and the acute infections including influenza are rendered much more dangerous by the presence of congenital heart disease.

(6) In cases which do not succumb to these complications there may be a gradual heart failure with oedema, comparable to that met with in the failing heart of acquired mitral disease.

On account of these complications a prognosis will always be a difficult matter.

Putting them aside, it must be admitted that even then the general prognosis for all cases is bad, for many die during the first two years of life.

Much importance must be attached to the symptoms. Paroxysms of dyspnœa, syncopal attacks, convulsions, or a persistently low temperature are serious whatever may be the physical signs. The social status too, has much influence on the prognosis.

Slight congenital lesions are compatible with life for many years, cases being on record that have survived the age of sixty.

As a general rule, if the lesions are of the usual type the outlook is better than when the case is unusual. A pulmonary stenosis for example, if of moderate degree, may reach adult life. Laurence Humphry is of opinion that in these cases the occurrence of a patent inter-ventricular septum renders the outlook more favourable.

Cyanosis may be present in cases which live for many years, and it is not a sign which can be relied upon in prognosis.

The supervention of gradual heart failure, with bronchitis, oedema, and tricuspid regurgitation, is a very serious matter, and some guide as to the probable occurrence of this can be obtained from the size of the heart and characters of the pulse and impulse. The change from a heaving right ventricle-beat to a feeble tapping one generally marks the last stage of the illness.

Treatment. Treatment is necessarily palliative.

Warm *clothing* and a warm equable climate are very necessary. The extremities should always be well covered, for these patients feel the cold severely. The *diet* must be digestible and judiciously arranged. No heavy meal should be permitted in the evening. Fat, in the form of cream, fat bacon, or cod liver oil, should be taken, for these cases, particularly children, are better when they are protected by a good layer of adipose tissue. Great precaution should be

taken to guard against the infectious diseases. In all cases in which there is a fair degree of health and strength, attention should be paid to education, and, as far as possible, steady exercise should be insisted upon. If the life is to be short, it should be happy; the spoiled invalid is a misery to himself and all concerned.

Drugs should be used sparingly. *Cod liver oil* and *malt*, combined sometimes with *iron*, is useful, and as a cardiac stimulant *strychnine* is generally preferable to *digitalis*.

For the fainting attacks, warm mustard baths and rapidly diffusible stimulants (6) are helpful in infancy. For older children, *strychnine* (5) should be given hypodermically under these circumstances.

When there is over-distension of the right ventricle the application of *leeches*, or for adults, *free venesection* may bring great relief. Gradual failure of the right side of the heart should be treated on the principles already indicated under chronic valvular disease.

CHAPTER XVI

ARTERIAL DISEASE AND ANEURYSM

Pathology of arterial disease—Arterio-sclerosis—Morbid anatomy—Aortitis—Morbid anatomy—Symptoms—Treatment—Atheroma—Causation—Morbid anatomy—Symptoms—Aneurysm of the thoracic Aorta—Causation—Morbid anatomy—Symptoms—Intra-pericardial aneurysm—Aneurysm of the first part of the arch—Of the transverse part—Of the descending part—Of the descending thoracic aorta—Diagnosis of aneurysm—Prognosis—Treatment.

ANEURYSM of the thoracic aorta and aortitis are conditions allied so closely to actual heart disease that an allusion to these lesions is essential. Unfortunately the pathology of aneurysm involves a consideration of the difficult subject of the pathology of arterial disease. An endeavour will be made in this chapter to explain some of the salient points in the general pathology of arterial disease and to illustrate their clinical importance by a short account of acute aortitis, atheroma of the aorta, and thoracic aneurysm. Under cardiac affections in renal disease the condition of the heart in the more general arterio-sclerosis has been already considered.

SALIENT POINTS IN THE PATHOLOGY OF ARTERIAL DISEASE

In arterial disease, as in disease of other organs, *acute*, *subacute*, and *chronic affections* occur. The type of an acute lesion is acute aortitis following upon a pyaemia or other

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severe infection ; syphilitic aortitis may produce a subacute lesion, and chronic disease is represented by atheroma. Again, arterial affections may be general in distribution as in the arterio-sclerosis of chronic renal disease, or focal, as in some cases of aneurysm of the arch of the aorta.

So much is clear, but difficulties arise when an attempt is made to define the exact causation of arterial disease, to classify accurately the morbid changes, and to interpret their exact meaning. Important as are the divisions of the wall of an artery into three coats, the intima, media, and adventitia, a clearer idea of the morbid processes is obtained from the conception of *an artery as a vital tube* nourished by small blood-vessels. These tubes must be smooth in bore, elastic, contractile, and of great strength. For these reasons they possess an endothelium, elastic fibres, muscle, and connective tissue.

The nourishment of the endothelium is attributed to the blood-stream circulating through the vessel, the rest of the wall is supplied by the vasa vasorum.

Three processes will need consideration in arterial disease :—

1. The morbid conditions producing the lesion.
2. The adaptive or compensatory changes.
3. The traumatic element. This is conveniently distinguished from the first or primary factor in arterial disease, in accordance with the generally received distinction between disease and traumatism.

1. The Morbid Conditions.

There are two definite possibilities. Bacterial and other poisons, or bacteria themselves, may be carried to the vessel-wall in the vasa-vasorum and thus produce lesions, or the

vasa-vasorum may themselves be damaged, their lumens occluded and the arterial wall thus starved of nourishment.

The particular tissue changes that will result in the vessel will be dependent upon the nature of the infection or poisoning, upon its virulence, and its duration. Doubtless also the result will depend in part upon the particular arterial constitution of the individual.

Whether the quality of the blood circulating within the arteries has any direct effect, apart from a thrombosis or embolism occurring within the vessel, is difficult to decide. The morbid lesion most easily understood is such an one as I found on one occasion in a case of fatal pneumonia. The first part of the aorta in this case showed *acute inflammation*, and in the subintimal tissue there were *pneumococci*. The endothelium was destroyed, the subintimal tissues were infiltrated with numerous leucocytes, and in places there was destruction of the intimal and muscular tissues. Here, then, was an acute process exactly comparable to an acute pneumococcal pleurisy or pericarditis. Such an example as this is, however, rare, and far more frequently the conditions that have to be studied *post mortem* are chronic, and this makes it most difficult to distinguish between the direct results of disease, the compensatory changes, and scarring processes.

There is one other acute process which can be recognized at the bedside, and this is *arterial spasm*. In some cases of chronic renal disease, apart from the persistent high blood-pressure, there are phases in which there is temporary arterial spasm. This certainly suggests a rapid accumulation in the circulation of deleterious substances which, circulating in the vasa-vasorum, produce an irritation of the middle coat of the vessels, or possibly stimulate the vaso-motor centre and cause vaso-constriction.

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Unfortunately our knowledge of the effects and duration of actions of bacterial toxins and other poisons upon the arterial tissues is very limited, and the problem of chronic arterial disease for this reason a very difficult one.

The *chronic arterial diseases* on this account are usually grouped under the general term of *arterio-sclerosis*.

The more localized form in which degenerative processes are conspicuous, and which is most frequently met with in the aorta and larger arteries, is distinguished by the name of *atheroma*. *Endarteritis obliterans* is applied to a condition in which there is striking proliferation of the inner coat without necrosis.

There are other descriptive terms in use for various forms of chronic arterial disease, some of them pathological and some clinical.

ARTERIO-SCLEROSIS

A. Pathological Classification

Type I. The primary change is apparently in the *muscular coat*. The muscular and elastic fibres undergo fatty degeneration and later calcification. The other coats are implicated but not essentially.

A feature of this condition is a yielding of the vessel-wall at the sites of disease with, as a result, the formation of small *pouches*. This process chiefly affects the peripheral arteries and is recognizable to the touch in the undue hardness of the radial artery.

This is sometimes termed the Moenekeburg type of arterio-sclerosis.

Type II. The marked changes occur in the *intima*, which is greatly increased in thickness. In some cases the

thickening is proliferative, in others there is fatty change and necrosis. The internal elastic lamina and the adjacent part of the muscular layer are damaged.

This type affecting the intimal and musculo-elastic layer is sometimes termed the Jores type of arterio-sclerosis.

Type III. The syphilitic type, in which there is a mes-arteritis; and as shown by Mott in the larger arteries, a great thickening of the vasa-vasorum and also intimal proliferation.

Josué, Fischer, Kurt, Klotz, and others have by intravenous injection of adrenalin, digalen and diphtherial toxins into rabbits produced arterial lesions comparable to Type I. Boinet and Romary, and Klotz, with the bacillus typhosus and streptococcus have with the same animals produced a condition comparable to Type II.

Klotz draws a sharp line of distinction between these two types: one produced by circulating toxins, the other by bacterial inoculations; but I am in agreement with Adami, believing that these may very possibly result from the same poisons acting in different degrees of virulence.

Without pursuing this pathological classification any further we can conclude that bacteria may themselves be carried by the vasa into the arterial wall, that many poisons circulating in the vasa of the vessel-wall may damage its tissues, and that arrest of the nutritive supply from disease of the vasa-vasorum themselves may produce necrotic change. Further, that all coats of the artery tend to suffer in disease whether this be acute or chronic, although in some cases the middle, and in others the intimal coat, shows the earliest signs. To what extent the blood circulating within the arterial channel influences the vessel-wall appears doubtful.

B. Clinical Classification

Clifford Allbutt classifies arterio-sclerosis from the clinical standpoint in three divisions:—

1. The toxic.
2. The hyperpietic.
3. The senile or involuntary.

Division 1. The toxic form is the result of various infections and poisons as, for example, syphilis, lead, and diabetes. This form *may or may not be associated with high blood-pressure.*

Division 2. The hyperpietic form is that in which the *blood-pressure is high from the first* and which he looks upon as the direct result of that high pressure. He points out that there may be a phase of the high pressure lasting for years before any arterio-sclerosis is evident.

Division 3. The senile or involutionary, which is characterized by great tortuosity and deformity of the vessels, without of necessity any increase in the blood-pressure at all, and which is independent of the blood-pressure.

If the reader will compare this classification with the pathological he will see that the former division is concerned entirely with toxic origin of arterio-sclerosis. If, however, we put the senile type of Allbutt into his toxic group, as he allows may be done, we have still the hyperpietic class in which he introduces this new factor into the problem—that *arterio-sclerosis* may be the *direct outcome* of the tensile strain of persistent and prolonged high blood-pressure.

This is the converse of the position adopted by Von Basch, who maintained that the arterio-sclerosis was a *cause and measure* of the high blood-pressure.

With all deference to these authorities, it appears to me that the occurrence of arterio-sclerosis is independent of the

blood-pressure, and that the hyperpietic type of arterio-sclerosis is toxic—the toxins in these cases having the power to produce high blood-pressure, as well as to damage the artery wall. Thus in chronic renal disease there is an irritation of the muscular fibres in the heart and all the blood-vessels. The result is an increase in the peripheral resistance, hypertrophy of the heart, and a rise of the blood-pressure, of toxic origin. There may be also some alteration in the capillaries or the tissues surrounding them tending to raise the peripheral resistance from the same cause, but this is more difficult to demonstrate, for we cannot detect their action, as in the arteries, by the hypertonus of the muscular coat. As the disease progresses the muscular coat is more than irritated, and becomes diseased, and finally arterio-sclerosis supervenes.

2. The Compensatory Changes

When the blood-pressure is high and the heart is hypertrophied there is a persistent strain upon the arteries which maintain the peripheral circulation in the face of an increased resistance. The muscular coat hypertrophies, and a condition arises termed by Savill *arterial hypermyotrophy*. This is an adaptive and compensatory change.

There is more discrepancy of opinion when the question of compensatory changes in the inner coat of the arteries is considered. The problem that arises is of this nature: examination of a circumscribed area of disease in the aorta shows partial destruction of the muscular coat and a hyperplasia of the intima in the weakened area. Is this hyperplasia compensatory or is it the result of disease? Thoma laid great stress upon the compensatory changes in the intima, but other authorities have questioned this view.

3. The Traumatic Element

Lastly, there is the *traumatic element* in arterial disease.

Sudden exertion raises the blood-pressure momentarily, and has sufficed to rupture the aorta. It is then only to be expected that when the arterial wall is diseased, arduous occupations involving sudden severe exertion will tend to produce a local yielding of the vessel and start an aneurysm.

ACUTE AND SUBACUTE AORTITIS

Acute aortitis is an occasional result of an acute infection attacking the vessel-wall. A septic infection may, for example, produce suppurative aortitis, and an acute syphilitic infection acute inflammation and ulceration.

Morbid anatomy. Raised gelatiniform pink patches are seen on the inner surface of the aorta and are almost invariably situated in the first two inches of the vessel.

The endothelium is swollen, and the intima is in parts necrotic, and all three coats of the vessel are infiltrated with leucocytes and themselves swollen. The vasa-vasorum are dilated and sometimes ruptured.

In some cases there are also acute changes in the myocardium as well as in the aorta.

Symptomatology. The symptoms are obscure: some *fever* at the onset, persistent, dull *substernal pain* over the region of the arch of the aorta, together with *angina pectoris*, are the most generally recognized. *Paroxysmal dyspnoea* may also occur. Among the physical signs there may be evidences of a *dilated aorta*, and in some cases the aortic valves are implicated and a *systolic* or *diastolic murmur* becomes audible. Or there may be a systolic murmur from dilatation of the aorta, the aortic valves themselves being

healthy. The condition is a grave one for several reasons ; the heart itself may be damaged by a similar process in the myocardium, the coronary arteries may be implicated, the aorta may be so weakened as to rupture, or aneurysmal dilatation or saccular aneurysm may directly follow the scarring of the lesions.

The treatment consists in absolute rest and a light diet. Pain is quieted, if severe, by opium, and if there is angina, this may be relieved by nitrites. Should syphilis be suspected, mercurials and the iodides should be pressed. Return to active life must be made with great caution, and particular attention given to the occurrence of substernal pain.

ATHEROMA

The causes may be divided into—

A. *Predisposing* ; and B. *Exciting*.

A. Among the predisposing are—

1. *Age*. Middle age and advanced life favour its occurrence.
2. *Sex*. Under fifty it is more frequent in males, over fifty the incidence is about equal in the two sexes.
3. *Heredity*. There is a decided family tendency to arterial degeneration.
4. *Occupation*. Occupations that are arduous and involve prolonged muscular exertion predispose to atheroma.

B. The *exciting causes* are not thoroughly understood. They may be provisionally grouped into—

1. *Toxic causes* associated with high blood-pressure, such as result from excess of nitrogenous food in those who are living a sedentary life. The gouty diathesis and lead-poisoning are examples of this group.
2. *Toxic causes*, not associated necessarily with high

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blood-pressure, e.g. syphilis, rheumatism, possibly alcohol and tobacco.

Many authorities believe that a persistent high blood-pressure will of itself produce atheroma.

Morbid Anatomy. The inner aspect of the vessel shows yellow elevations dull in colour and smooth on the surface. These vary in size and shape, and in the later stages are ulcerated. Further there is often calcification which may be extreme. The convexity of the arch of the aorta is most frequently damaged. It is a natural thought to look upon calcification as a very chronic process, but experimental investigations point to its occurrence as consistent with quite recent changes. The intima and musculo-elastic layer of the media are particularly damaged, and in the intima there are necrotic patches. The vasa-vasorum may be obliterated by endarteritis obliterans.

Whether the changes in the arterial wall are entirely secondary to the impoverished blood supply from primary vasal disease or whether they are in part primary and the result of toxæmia or strain is debated.

The symptoms are not characteristic, there may or may not be general arterio-sclerosis. In some cases there is general dilatation of the arch of the aorta giving rise to dulness over the manubrium sterni, and a definite shadow on the fluorescent screen, and a low-pitched ringing second sound over the aortic cartilage. If the aortic valves are implicated, there will be stenosis or regurgitation or the combined lesions. If the coronary arteries suffer there may be the symptoms of coronary disease.

ANEURYSM OF THE AORTA

An *aneurysm* is a pulsating tumour, the result of the giving way of the wall of an artery weakened by disease or injury. If the aorta is generally dilated the condition is one of *fusiform aneurysm*, if the dilatation is localized to one area of the circumference, a *saccular aneurysm*. Occasionally the inner part of the arterial wall gives way, but the outer holds firm, and the blood tears up the middle coat, making its way back again at some more or less distant spot into the lumen of the vessel: this is termed a *dissecting aneurysm*.

Ætiology. The *predisposing causes* are middle and advanced age, the male sex, and arduous employments, particularly those which call for sudden and severe intermittent exertion.

The *exciting causes* are those which produce local disease of the aorta, notably syphilis, and probably, as the Broadbents maintain, other bacterial infections, and possibly also toxins not necessarily bacterial. Sudden variations in the blood-pressure may further act as a determining cause when the arterial wall has been weakened by disease. Aneurysm in malignant endocarditis has been considered earlier.

Morbid Anatomy. This particular form of arterial disease has, in addition to its local distribution, a *subacute character*. It is not severe enough to cause rupture of the aorta, and not sufficiently chronic to allow reparative processes to come step by step to the rescue, but it is sufficiently acute and deep-seated to damage grievously the muscular coat, and to prevent the reparative keeping pace with the destructive processes.

Post-mortem examination of a commencing aneurysm shows that the intima not only is damaged but the *middle coat* also is much thinned. In syphilis the process commences as a

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mesarteritis. As the aneurysm increases in size the inner and middle coats are practically destroyed, but the adventitial layer increases in thickness and forms the main sac. In the saccular form of aneurysm the opening from the aorta varies considerably in extent, the smaller it is the more probable that some coagulation of the blood will occur in the aneurysm owing to the stasis of the circulation. This process of blood-clotting is an important event in the history of the disease, for not only does it serve to strengthen the sac but it may sometimes bring about a *spontaneous cure* by obliteration of the cavity.

The wall of the aneurysm comes into contact with surrounding structures and incorporates them into itself by a process of traumatic inflammation, thus often prolonging the duration of life. Eventually, however, the general rule is that the aneurysm gives way, usually at some dependent or unsupported spot, and *fatal haemorrhage* results.

Symptomatology. When *aneurysm* is considered as a clinical disease, it is apparent that it is essentially a tumour which tends to enlarge and cause persistent pressure. This tumour from its pulsatile character has a peculiar power of erosion. The primary cause of the arterial disease now becomes secondary in importance to the result it has produced.

The symptoms are accordingly pressure symptoms, and the physical signs those of a tumour connected with an artery.

Both the symptoms and physical signs will differ with the particular region of the thoracic aorta affected.

These aneurysms may be divided conveniently into—

1. Intra-pericardial aneurysm.
2. Aneurysm of the ascending part of the arch.
3. Aneurysm of the transverse part of the arch.

4. Aneurysm of the descending part of the arch.

5. Aneurysm of the descending thoracic aorta.

1. **Intra-pericardial aneurysm.** This generally occurs at the site of one of the sinuses of Valsalva, and seldom reaches any large size.

It is easily overlooked, or may give rise to no trouble at all before a fatal rupture occurs. **The cardinal signs are :—**

(1) Angina pectoris.

(2) Aortic incompetence.

(3) Impairment of percussion note to the right of the sternum in the second right intercostal space.

(4) Pressure on the superior vena cava.

Rupture generally takes place into the pericardium, sometimes into the superior vena cava or pulmonary artery.

(a) *Intra-pericardial rupture* need not be instantly fatal if the leak is very small. In such cases there is precordial pain and distress, with cardiac excitement, and possibly pericardial friction. When the haemorrhage is large, death is almost instantaneous.

(b) *Rupture into the superior vena cava* is compatible with survival for some months. This depends again upon the extent of the rupture. *The symptoms* are, a sudden, tense, bursting sensation in the head, with the rapid onset of extreme oedema of the face and neck, dyspnoea and cyanosis. The jugular veins are distended and thrombosis may follow. With the establishment of collateral circulation the veins over the front of the chest enlarge. A *loud, continuous, humming* murmur may appear, together with a thrill in the aortic region. The great obstruction to the venous circulation in the brain may cause severe mental symptoms.

(c) *Rupture into the pulmonary artery.* The symptoms are sudden pain in the chest, with breathlessness. The characteristic sign is a murmur, comparable in some respects to that found in cases of patent ductus arteriosus. It is in character a continuous, loud, harsh sound, rising and falling with systole and diastole, with a maximum intensity in the third intercostal space on the left side.

These patients may live for months. There usually follow pulmonary incompetence, right heart failure, and dropsy.

2. Aneurysm of the first part of the arch, termed by Sir W. Broadbent 'the aneurysm of signs'. The most prominent symptom is pain due to pressure of the sac upon the intercostal nerves. This may also be referred down the inner side of the right arm to the elbow. The physical signs are:—

1. *Pulsation* in the second intercostal space on the right side.

2. On *palpation* the pulsation may be felt even before it is visible, and in addition a diastolic shock may be detected.

3. There is *abnormal dulness* to percussion in the second and third intercostal spaces.

4. The *aortic second sound* is low-pitched and ringing, and there may be a systolic murmur.

This aneurysm may reach a *great size*, usually extending outward to the right, and forward. The ribs in front may be eroded, and a large tumour may appear under the skin, which, however, is itself very rarely ulcerated.

It may press upon the *right auricle* and *superior vena cava*, producing great dilatation of the tributary veins, and the gradual establishment of a collateral circulation. It is in these cases that the condition simulates *mediastinal tumour*.

Again, it may press upon the *right bronchus*.

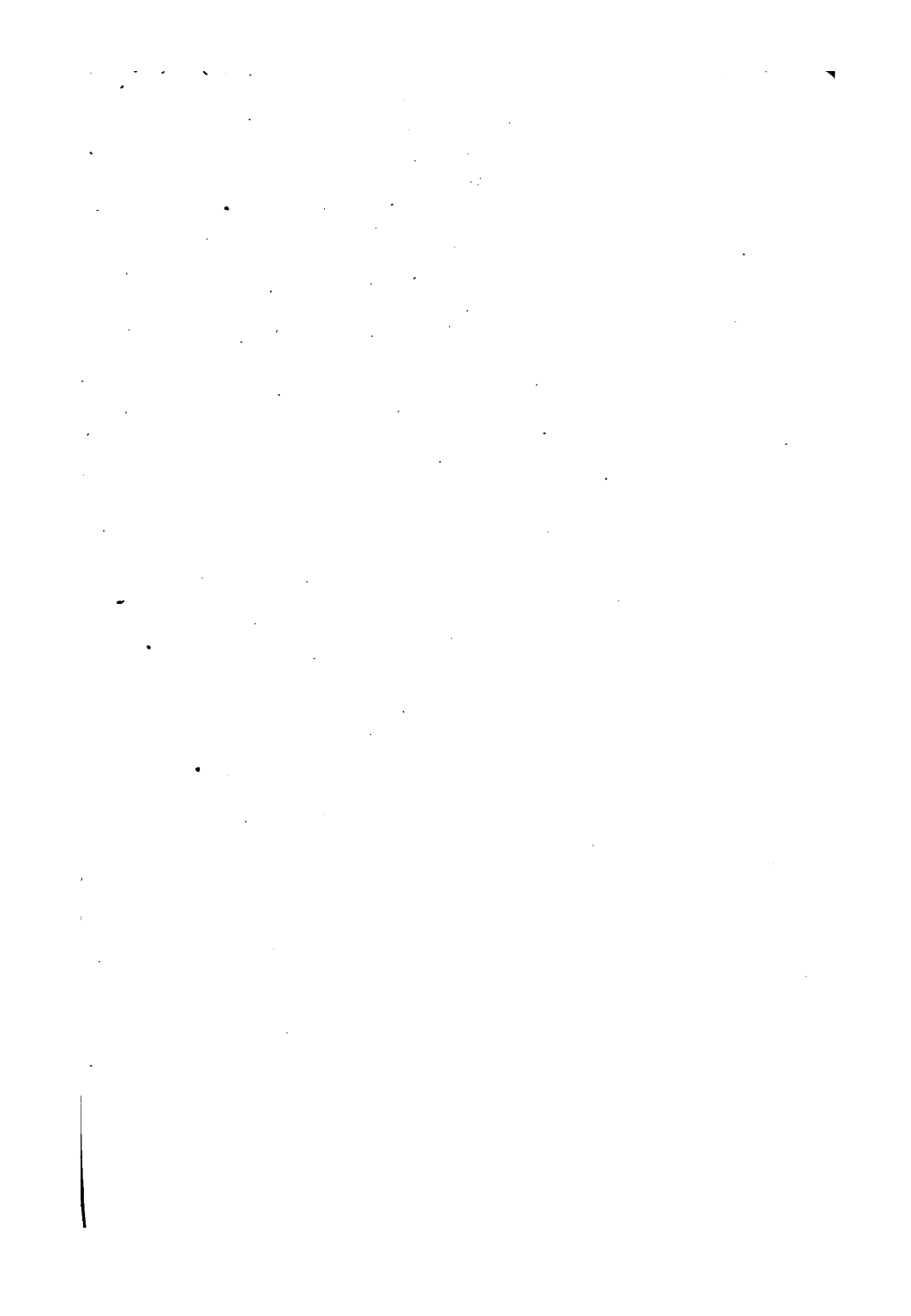


PLATE XVI



Aneurysm of the transverse part of the arch of the aorta, illustrating the involvement of the large arterial branches in the sac, and pressure upon the bifurcation of the trachea.

*From a specimen in the Museum of University
College Hospital.*

This at first abolishes the vesicular murmur over the upper lobe of the right lung, although the percussion note may be at first unaltered. The signs are tersely expressed by Sir W. Broadbent as those of '*resonance and silence*'. Later there is pulmonary collapse with impairment of the note, tubular breathing and adventitious sounds.

Rupture occurs most often into the pericardium at the lowest limit of the sac, in the angle between the superior vena cava and aorta; sometimes into the pleura, and sometimes into the lung.

When it occurs into the pleura, if the patient survives, there are signs of a right pleural effusion, and it may happen, to one's great discomfiture, that an exploring syringe-needle draws off *pure blood*.

If there is repeated *haemoptysis* and the physical signs in the lungs from pressure are conspicuous, tuberculosis may be diagnosed.

Some of these cases of aneurysm run a long course.

3. **Aneurysm of the transverse part of the arch of the aorta**, termed by Sir W. Broadbent 'the aneurysm of symptoms'. The reader by referring to the anatomy of the aorta (Chapter i, p. 9), will appreciate the symptoms the more easily.

This aneurysm appears clinically under many different guises.

1. *Alteration in the voice and brassy cough*. The *left recurrent laryngeal nerve* as it winds round the aorta, is frequently implicated by an aneurysm; but the right nerve, winding round the right subclavian artery, is not so often involved.

The result of pressure upon the left recurrent laryngeal nerve is to produce abductor paralysis of the left vocal cord, which becomes fixed in the cadaveric position; the

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free excursion of the other cord protects against any serious dyspnoea, but there is some breathlessness on exertion. The voice and cough are altered and brassy in timbre when there is pressure on the trachea or a main bronchus. An irritant cough is an early and frequent sign of aneurysm in this position.

2. *Dyspnoea.* There may be persistent dyspnoea from pressure upon the trachea or main bronchi, and in addition



FIG. 10. *a.* Right radial. *b.* Left radial. From a case of large thoracic aneurysm. The tracings were taken at equal pressures (T. Lewis).

terrible paroxysms of difficult breathing, due to sudden accesses of this pressure or more rarely to double abductor palsy.

3. The *tracheal tug* has been described on page 32.

In some cases the trachea is pushed on one side.

4. *Alteration in the pulses.* It is in aneurysm of the transverse arch that this valuable sign is most often discovered. It may be a very early manifestation. The usual result is to find that the left radial artery has a smaller pulse-wave, longer in duration, and detected somewhat later than in the right pulse. In some cases the right pulse is affected by implication of the innominate artery. Again, one radial or one carotid pulse may not be appreciable to the finger at all.

PLATE XVII



I

Radiogram of heart, taken from the back. (A) points to a shadow of the thoracic aorta as it deviates to the left of the middle line. This may be mistaken for an aneurysm. (Higham Cooper.)



II

Radiogram illustrating a large aortic aneurysm. The photograph is taken from the front. Below, and to the right, the upper part of the heart shows as a dark shadow, and above it rises the fainter ovoid swelling of the aneurysm. (Higham Cooper.)

The *explanations* are various.

In some instances there is interposed between the innominate artery and left subclavian a very large aneurysm in which all the resilience of the normal vessel is wanting; in other cases, the tumour presses on one or other vessel; in others, the atheromatous process implicates the origin of one of the vessels.

5. By no means a frequent symptom, but a very important one, is *pressure on the oesophagus*. This is particularly liable to occur with small aneurysms, springing from the posterior part of the arch and pinning the oesophagus against the vertebral column, and it may be the only symptom. Enormous aneurysms may fail to cause dysphagia, the oesophagus eluding them in a remarkable way; and although the well-known rule in suspected stricture of the oesophagus should be obeyed and no bougie passed until aneurysm has been looked for, the physician may be defeated, and not detect this small aneurysm.

Rupture into the oesophagus may prove rapidly fatal by haematemesis.

6. *Pressure on the left bronchus* will abolish the vesicular murmur on the left side and later give rise to collapse of one or both of the lobes of the lung.

7. *Pressure upon the left innominate vein* will produce fullness of the left external jugular and enlargement of the veins on the surface of left side of the chest.

8. *Pressure upon the sympathetic*. In an early stage the pupil may be widely dilated, at a later stage it is contracted. In some cases there is also unilateral flushing of the face and sweating.

Wall, Ainley Walker and others have attributed the alteration in the pupils in aneurysm to inequality of blood-pressure in the ophthalmic arteries, the result of ab-

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normal vascular conditions, rather than to pressure on the sympathetic.

9. *Pressure on the thoracic duct* is associated with profound wasting.

10. *Pressure on the deep cardiac plexus* may be a cause of palpitation and probably of sudden death.

There are some important physical signs.

Among these, marked dulness over the manubrium sterni, and to its immediate right and left; undue pulsation immediately above the manubrium. In some cases the sternum is eroded and a small tumour pushes its way through. A systolic murmur may also be audible over the aneurysm and a diastolic shock be present.

4. Aneurysm of the Descending Part of the Arch

This is often the most difficult to detect and for this reason the fluorescent screen is a great assistance. There may be pressure on the *oesophagus*, or the *root of the left lung*, or the *left recurrent laryngeal nerve*. *Severe pain* radiating round the left side of the chest, extending down the left arm, should suggest erosion of the left side of the bodies of the fourth dorsal and adjacent vertebrae, and pressure on the intercostal nerve roots.

In some cases on auscultation a *systolic murmur* may be audible over the site of the aneurysm and yet over the aortic valve no murmur be heard. This appearance of a murmur along the course of the vessel is certainly suggestive of an aneurysm.

5. Aneurysm of the Descending Thoracic Aorta

This again is often most difficult to detect. *Pain* severe and lancinating in character is the most usual symptom. There may be no other evidence, and for this reason the

fluorescent screen is again a most valuable aid. Occasionally there is *dysphagia* from pressure on the *oesophagus*. The erosion of the dorsal vertebrae and the adjacent parts of the left ribs, with implication of the spinal root, accounts for the pain, and naturally suggests caries of the spine. The back of the left side of the chest should be most carefully examined in a good light, and in some cases a *slight but definite pulsation* will be evident. When the aneurysm also implicates the upper part of the abdominal aorta, pain may be referred to the *left kidney*, which organ has on more than one occasion been explored for stone when the true explanation was an aneurysm. The supervention of *sudden paraplegia* suggests rupture into the spinal canal.

Diagnosis of Aortic Aneurysm

Diagnosis. It is a medical aphorism that a healthy-looking male adult who comes to a doctor complaining of persistent cough and pain in the chest should be examined with a view to the presence of thoracic aneurysm.

When the veins are enlarged a *mediastinal tumour* is suggested, but in this condition there is no pulsation unless the tumour is one of the rare pulsating sarcomata. There are no cardiac murmurs. There may be a general lifting of the chest when the tumours push forward the heart, but there is an absence of the diastolic shock.

Tuberculosis with profuse haemoptysis may be distinguished by the presence of tubercle bacilli in the sputum, and the absence of a tumour, but sometimes the difficulty is very great and the screen necessary. *Fibrosis of the lungs* and *malignant growths* of the lungs may simulate aneurysm, as also the rare condition of *pulsating empyema*.

Aneurysm may be mistaken for *malignant stricture of the oesophagus*, or the reverse mistake may be made. *Spinal*

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curies or *cancer of the spine* may easily be mistaken, but the screen will be again a useful assistance.

The valvular lesion most likely to be mistaken is *aortic regurgitation* with marked enlargement and pulsation of the aorta. The physical signs must be carefully reviewed. Great enlargement and hypertrophy of the heart strongly favours aortic regurgitation, for it is exceptional, as Calvert showed, to find that an aneurysm causes considerable enlargement of the heart.

Prognosis in Aortic Aneurysm.

The prognosis is always grave and very uncertain. The duration of life may, however, be in exceptional cases as long as ten or fifteen years; and I have made a *post-mortem* examination upon a middle-aged man who died from malignant disease, and found a completely cured aneurysm at the commencement of the left subclavian artery the size of a fowl's egg.

When the tumour can be defined, symptoms of increasing pressure are bad; diminution in size, pulsation, and pain are good. Haemorrhage or slight oozing from any region implicated is a warning sign, but a very profuse haemorrhage—almost to death—has been followed by extraordinary improvement. Death frequently occurs from causes other than rupture.

Intra-pericardial aneurysm ruptures early.

Aneurysm of the first part of the arch may, if the sac comes forward, last many years. Backward extension of the sac is less favourable. The secondary results of aneurysm of the transverse part of the arch add considerably to the dangers.

Treatment of Aortic Aneurysm.

Treatment. This is unsatisfactory.

The method which still appears to be the most advocated is prolonged *rest* in the recumbent position, restriction of fluids to the minimum the patient can tolerate, and light meals in which meat is curtailed.

Together with this, *iodide of potassium* is given in large doses three times a day.

These doses may vary between 10 and 30 grains at a time. Should iodide of potassium be ill borne *iodalbin* in ten-grain doses may be substituted.

It is not possible in a great many cases for the patient to be content or able to carry out absolute rest, and some prefer the risk of an ordinary life to the bare possibility of a cure by this method. In such cases no violent exercise is permitted, the food is kept small in bulk, and fluids scanty. The iodides are persevered with, and the patient warned against the danger of sudden exertion, sexual intercourse, and the taking of alcohol. When there is much pain, the relief produced by complete rest will be the strongest incentive to the patient to persevere. In all cases of aneurysm *the bowels should be kept well open.*

Lancereaux has introduced a form of treatment by the subcutaneous injection of *a saline gelatin solution*. The gelatine is believed to be absorbed and to increase the coagulating power of the blood.

The occurrence of tetanus after these injections, which was learnt by bitter experience, can be corrected by efficient sterilization and need not be taken into account now.

It is unfortunately a treatment which causes great pain.

The injection is made into the buttock. A 2% solution of gelatine is made in normal salt solution, and carefully

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sterilized. Three ounces are injected at a time, and the injections are made every other day. In this country the reports have not been encouraging.

Calcium chloride in ten-grain doses is believed to increase the coagulability of the blood, but has been in my experience very disappointing.

Pain in aneurysm will frequently need the use of *opium* or *morphia*. A great urgency in treatment is intense paroxysmal dyspnoea. There is in these cases, generally, pressure upon the lower end of the trachea or a main bronchus, so that *tracheotomy* can be dismissed as purposeless. *Venesection* may bring great relief, but cannot be frequently repeated. *Anaesthesia* is also helpful, and sometimes *nitrite of amyl* relieves the pain.

APPENDIX

I. PRESCRIPTIONS FOR CARDIAC DISEASE.

For an adult.

1. R Sodii Salicylatis gr. x
Sodii Bicarbonatis gr. x
Tinct. Nucis Vomicae ℥v
Tinct. Lavandulae Co. ℥ss.
Glycerini ℥ss.
Aquae ℥i

Fiat Mist.

S. Two tablespoonfuls every four hours until the pain is relieved, then every six hours.

2. R Pot. Bicarbonatis gr. xx
Sodii Bicarbonatis gr. x
Aquae ℥i

Fiat Mist. Alkalina.

- R Quinae Sulphatis gr. ij
Acidi Citrici gr. xvij
Syrupi Limonis ℥ss.
Aquae ℥i

Fiat Mist. Acida.

S. Two tablespoonfuls of each mixture to be taken together as a draught every six hours.

For a child aet. 7.

- | | |
|-----------------------|-------|
| Salicini | gr. x |
| Sodii Bicarbonatis | gr. v |
| Tinct. Nucis Vomicae | ℥ij |
| Tinct. Lavandulae Co. | ℥xv |
| Glycerini | ℥xv |
| Aquae | ℥ij |

Fiat Mist.

S. Two teaspoonfuls every four hours, then every six hours.

- | | |
|-----------------------|-------------------|
| Quinae Sulph. | gr. $\frac{1}{2}$ |
| Ammon. Carb. | gr. i |
| Pot. Bicarb. | gr. v |
| Mucilag. Tragacanthae | ℥i |
| Syrupi Aurantii | ℥ss. |
| Aq. Chloroform. | ℥ij |

Fiat Mist.

S. Two teaspoonfuls every six hours.

3. R Sodii Bromidi	gr. xx	Sodii Bromidi	gr. x
Nepenthe	℥ viii	Nepenthe	℥ iiij
Tinct. Lupuli	℥ i	Tinct. Lupuli	℥ xx
Aquae Chloroformi	℥ i	Aquae Chloroformi	℥ ij
Fiat Mist.		Fiat Mist.	

S. Two tablespoonfuls at night and as directed.

S. Two teaspoonfuls at night and as directed.

4. R Paraldehyde	℥ i
Tinct. Aurantii	℥ i
Syrupi	℥ i
Aquae Chlor.	℥ i
Fiat Mist.	

S. To be taken one hour after the last meal.

5. R (a) Tinct. Digitalis	℥ vij	(a) Tinct. Digitalis	℥ iv
or Tinct. Strophanthi	℥ x	Liq. Strychninae	℥ i
Liq. Strychninae	℥ iiij	Sp. Chloroformi	℥ v
Sp. Chloroformi	℥ x	Aquae	℥ ij
Aquae	℥ i	Fiat Mist.	

Fiat Mist.

S. Two tablespoonfuls every six hours.

S. Two teaspoonfuls every six hours.

(b) Hypodermically
Liq. Strychninae hydrochlor. 1 % Solution
S. ℥ iiij 6tis horis.

(b) Hypodermically
Liq. Strychninae hydrochlor. 1-400 Solution
S. ℥ iv 6tis horis.

6. R Spiritus Aetheris	℥ ss.
Spiritus Ammoniae aromatici	℥ ss.
Tinct. Aurantii	℥ ss.
Aquae Camphorae	℥ ss.

Fiat Mist.

S. Every two hours as required.

Half the adult dose.

PRESCRIPTIONS FOR CARDIAC DISEASE 295

7. R. Ferri et Quininae

Citratis	gr. x	
Liq. Arsenicalis	℥ iij	
Sodae Bicarb.	gr. x	Half the adult dose.
Syrupi Aurantii	℥ ss.	
Infusi Calumbae	℥ i	

Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

Some types of aperients for patients suffering from Cardiac weakness, whether valvular, myocardial, or the result of Anaemia.

A dinner pill for habitual use.

Where Anaemia is marked.

8. R. Ext. Aloes	gr. i	Ext. Nucis Vomicae	gr. ss.
Ext. Nucis Vomicae	gr. ss.	Ext. Cascarae Sagradae	gr. ij
Pulv. Ipecac.	gr. ss.	Ferri Sulph. exsicc.	gr. i
Quinae Sulphatis	gr. i	Confectio Rosae	q. s.
Saponis	gr. ss.	M. Fiat pil.	

Misce : fiat pil.

S. To be taken immediately before dinner.

S. One three times a day.

Some patients are better suited by salines, and can take, fasting, three or more ounces of Apenta, Friedrichshall, or Rubinat water, or two drachms of effervescing sodium phosphate solution.

When the liver is sluggish from an impaired circulation.

R. Podophyllini	gr. $\frac{1}{4}$
Aloes Barb.	gr. i
Capsici	gr. ss.
Ext. Belladonnae Vir.	gr. $\frac{1}{4}$
Ext. Gentianae	q. s.

Fiat pil.

S. One or two in the day.

*For an adult.**For a child aet. 7.*

9. R. Tinct. Digitalis ℥ij
 Caffeinae Citratis gr. v
 Sp. Chloroformi ℥x
 Syrupi Aurantii ʒss
 Aquae ʒi

Half the adult dose.

Fiat Mist.

S. Two tablespoonfuls every four hours.

10. R. Tinct. Digitalis ℥ij
 Liq. Trinitrini ℥ij
 Sp. Chloroformi ℥x
 Syrupi Aurantii ʒss.
 Aquae ʒi

Fiat Mist.

S. Two tablespoonfuls every six hours.

11. R. Bismuthi Carb. gr. x
 Sodae Bicarb. gr. x
 Sp. Chloroformi ℥x
 Inf. Caryophylli re-
 centis ʒi

Half dose.

Fiat Mist.

S. Two tablespoonfuls after meals.

- (b) Listerini ʒss.
 Tinct. Zingiberis ℥xxx
 Tinct. Cardamomi Co. ʒi
 Aq. Menth. Pip. ʒi

Half dose.

Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

12. R. Liq. Morphinae hydro- Tinct. Camph. Co. ℥v
 chloratis ℥ij Ext. Glycyrrhizae liq. ℥xx
 Sp. Chloroformi ℥ij Aquae ʒi
 Glycerini } a-a ʒi
 Aquae }
 Fiat Mist.

S. A teaspoonful when the attack is severe.

Fiat Mist.

S. A teaspoonful when the attack is severe.

PRESCRIPTIONS FOR CARDIAC DISEASE 297

13. R. Tinct. Nucis Vomicae ℥ viij
 Ammonii Carbonatis gr. iv
 Liq. Arsenicalis ℥ iij Half dose with Syrup, vice the
 Tinct. Cardamomi Co. ʒ ss. cardamoms.
 Aquae Chlor. ʒ i

Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

14. R. Tinct. Valerianae Am-
 moniatae ʒ ss.
 Ammonii Bromidi gr. xx
 Aq. Camphorae ʒ i

Fiat Mist.

S. Two tablespoonfuls every four hours.

15. R. Tinct. Ferri perchlor. ℥ x Sodii formatis gr. viij
 Liq. Arsenici hydro- Mucilag. Tragacanthae q. s.
 chlorici ℥ iij Aquae Chlor. ʒ ij
 Liq. Strychninae ℥ iij Fiat Mist.
 Glycerini ʒ ss.
 Syrupi Aurantii ʒ ss. S. Two teaspoonfuls three times
 Aquae Chloroformi ʒ i a day after meals.

Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

16. R. Liq. Atropinae Sulphatis ℥ i
 Liq. Strychninae hydro-
 chloratis ℥ i
 Syrupi Aurantii ℥ x
 Aquae Chloroformi ʒ i
 Fiat Mist.

S. A teaspoonful every four hours as directed.

(Pharm. Hospital for Sick Children, Great Ormond Street.)

17. R. Liq. Ferri perchlor. ℥ v
 Tinct. Nucis Vomicae ℥ ij
 Syrupi Aurantii ℥ xx
 Aquae Chlor. ʒ ij

Fiat Mist.

S. Two teaspoonfuls after meals.

18. R. Liq. Strychnini hy-
drochloratis ℥ iij
Acidi Nitrici dil. ℥ x
Tinct. Chlor. Co. ℥ x
Inf. Gentianae Co. ℥ i

Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

19. R. Caffeinae gr. ij
Sodii Benzoatis gr. ij
Syrupi Floris Au-
rantii ℥ xv
Aquae Chlor. ℥ ss.

Fiat Mist.

S. A tablespoonful every four hours.

20. R. Liq. Hydrargyri per-
chloridi ℥ i
¹ Potassii Iodidi gr. v
Infus. Gentianae Co. ℥ i

Fiat Mist.

S. Two tablespoonfuls every four hours.

21. R. Ferri et Quininae
Citratis gr. x
Liq. Strychninae ℥ iij
Glycerini ℥ ss.
Tinct. Aurantii ℥ ss.
Infus. Aurantii Co. ℥ i

Fiat Mist.

S. Two tablespoonfuls three times a day after meals. S. Two teaspoonfuls for a child aet. 7, three times a day.

¹ The Iodide should be pushed.

PRESCRIPTIONS FOR CARDIAC DISEASE 299

Some Cardiac Tonics which may succeed where Digitalis fails.

22. R (a) Tinct. Strophanthi ℥ x

Liq. Ammon. Acetatis ℥ iv

Tinct. Nucis Vomicae ℥ vij

Aq. Menth. Pip. ℥ i

Fiat Mist.

S. Two tablespoonfuls every six hours. S. Three teaspoonfuls for a child aet. 7.

(Dilated Heart and Renal Disease.)

R (b) Tinct. Convallariae ℥ xv

Potassii Acetatis ℥ ss.

Magnesii Sulphatis ℥ i

Ammonii Carbonatis gr. v

Infus. Calumbae ℥ i

Fiat Mist.

S. Two tablespoonfuls every four hours.

23. R Pulv. Digitalis gr. i

Pulv. Scillae gr. ij

Pil. Hydrargyri gr. v

Fiat Pil.

S. One, morning and evening.

24. R Sp. Ammoniae Aromat-

tici ℥ i

Sodii Bicarbonatis gr. x

Liq. Trinitrini ℥ i

Tinct. Cardamomi Co. ℥ i

Aquae ℥ iss.

M. F. haustus.

To be slowly sipped at the first warning of a paroxysm (Powell).

25. R Liq. Arsenicalis ℥ iij

Ammon. Carb. gr. iv

Tinct. Card. Co. ℥ i

Inf. Gentianae Co. ℥ i

Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

26. R Potassii Iodidi gr. v
 Ammonii Bromidi gr. x
 Tinct. Belladonnae (1) v
 Syrupi Aurantii ʒss.
 Aquae Menth. Pip. ʒi
 Fiat Mist.

S. Two tablespoonfuls three times a day.

27. R Pot. Bicarb. gr. x
 Liq. Arsenicalis (1) iij
 Tinct. Nucis Vomicae (1) vj
 Aquae Chloroformi ʒi
 Fiat Mist.

S. Two tablespoonfuls three times a day after meals.

II. SOME DIETS IN CARDIAC DISEASES

Dietetic rules for elderly patients with Myocardial weakness.

Balfour.

1. There must not be less than five hours between the meals.
2. No solid food between meals. Hot water sipped three hours after a meal will rouse the flagging digestion.
3. The most important meal should be in the middle of the day.
4. The meals should be as dry as possible, and the bulk of fluid never exceed 5 oz.

Diet (1). *Type diet for a weak heart.*

Balfour.

Breakfast, 8.30. One slice of dry toast.
 One soft-boiled or poached egg, or some white fish.
 5 oz. of tea or coffee with cream and sugar, or infusion of cocoa nibs.

SOME DIETS IN CARDIAC DISEASES 301

Dinner, 1.30.

Two courses.
Fish and meat,
or
Fish and pudding,
or
Meat and pudding.

Soups, pastry, pickles, and cheese forbidden.

Tea, 5 p.m. 3 to 4 oz. tea infused not more than four minutes.

Supper, 7 p.m. White fish and potato, or beef extract. Milk pudding.

Diet (2). *The diet de rigueur for a case of Anasarca. Balfour.*

Breakfast. One slice of dry toast, and one cup of tea with sugar and cream.

Dinner. The lean of two chops or its equivalent, dry toast, and half an ounce of brandy or whisky in two ounces of water.

Supper. As much dry toast as desired, and half an ounce of stimulant.

Diet (3). *Some points in the dietary of patients suffering from Myocardial weakness due to fatty infiltration.*

Animal food should be taken in moderate amount, and consist of the lean of meat, game, and poultry. *Fish*, avoiding salmon and other fatty kinds, is permitted. A small quantity of *fat* should be taken for health's sake. *Farinaceous and starchy* food are reduced, and no sugar allowed. About five ounces of fluid is the limit with the meals, and hot water should be taken between them. Fresh vegetables and fruit are allowed.

In some cases of much difficulty, met with among fat anaemic women, an *exclusive milk diet* with rest and massage may be necessary at first.

Diet (4). *Some points in the dietary of the Gouty.*

There is no single diet suitable for the gouty, and here only a few points upon which there is general agreement are given.

Food should be plainly cooked. Stewed and fried dishes are better avoided, and roasting and grilling preferred. *Browned fats* and *oily fishes* are unsuitable, but such fats as bacon, butter, and suet can be digested. *Pastry*, rich *saucers*, and *sweets* are contra-indicated. Poultry and tender game, oysters, lobsters, and crabs are generally allowable. Some wine may be taken with the meals, in small quantities.

Diet (5). *Some points in the diet of Cardiac weakness with Anaemia.*

The *nitrogenous* elements are of first importance, and milk is of great value in the early stages of treatment.

Fats are also indicated in the form of butter, cod-liver oil, cream, or fat bacon.

Plenty of salt and green vegetables should be permitted.

Breakfast should be taken in bed.

III. THE SCHOTT MOVEMENTS

1. *Arms extended* in front of the body, at the level of the shoulders, and palms touching. The patient slowly moves them outward until in a line with one another, against the gentle resistance of the operator. This done, they are slowly brought back to the original position.

2. *Arms dependent*. Patient flexes forearm until hand touches shoulder, and then returns to original position. One forearm is flexed at a time.

3. *Arms dependent*, then slowly swept round in the arc of a circle until the thumbs touch above the head; then back again.

4. *Arms dependent*. Hands meeting over abdomen, and the first phalanges pressed together against one another. In this position the arms are raised to the level of the head, and then returned to the same position.

5. *Arms at attention*, then slowly raised forward to the vertical position and back again.

6. *The same movements* as No. 1, with the fist clenched.

7. The same as No. 2, but with the fist clenched.

8. *Flexion of the trunk* to a right angle, without flexion of the knees, and return to the original position.

9. *In the erect position* rotate trunk without moving the feet, first to one side, then to the other, then back to original position. The operator will place one hand in front of the advancing shoulder, the other behind the retreating one.

10. *In the erect position.* Flex the trunk laterally, first to one side, then to the other, and return.

11. *Erect position.* Hands dependent, arms extended. Make a complete revolution of first one arm, then the other.

12. *Erect position.* Arms extended, hands against thighs. Move upward and backward without flexion of the trunk, and return to original position.

13. *Erect position.* Stand supported by hand on a chair. Flex thigh to abdomen, and back again.

14. *Same position.* Keep the leg stiff, and bend the whole limb first forward, and then backward.

15. *Erect position.* Flex and extend leg on thigh.

16. *Erect position.* Abduction and adduction of the extended lower extremity.

17. *Erect position.* Arms abducted to the horizontal line, rotated forward and backward to their extreme limits.

18. Flexion and extension of the wrist.

19. Flexion and extension of the ankle.

All these movements are made slowly against the slight resistance of a skilled assistant. The movement is repeated, and rest is allowed between each of them. The condition of the patient is carefully watched during the exercises.

ARTIFICIAL NAUHEIM BATHS

1. *The Weak Bath.*

To 10 gallons of water at 95° F. add 1 lb. of Sodium Chloride, 1½ oz. of Calcium Chloride; increase until 3 lb. of Sodium Chloride and 4½ oz. of Calcium Chloride are used.

Duration of bath, at first, five minutes; later, fifteen.

Temperature lowered from 95° F. to 85° F.

2. The Strong Bath (Efferrescing).

To 10 gallons of water add Sodium Chloride 3 lb., Calcium Chloride $4\frac{1}{2}$ oz., Sodium Bicarbonate 2 oz., lastly 3 oz. of Hydrochloric Acid. Increase the strength until the proportions are—8 oz. of Alkali, 12 oz. of Acid.

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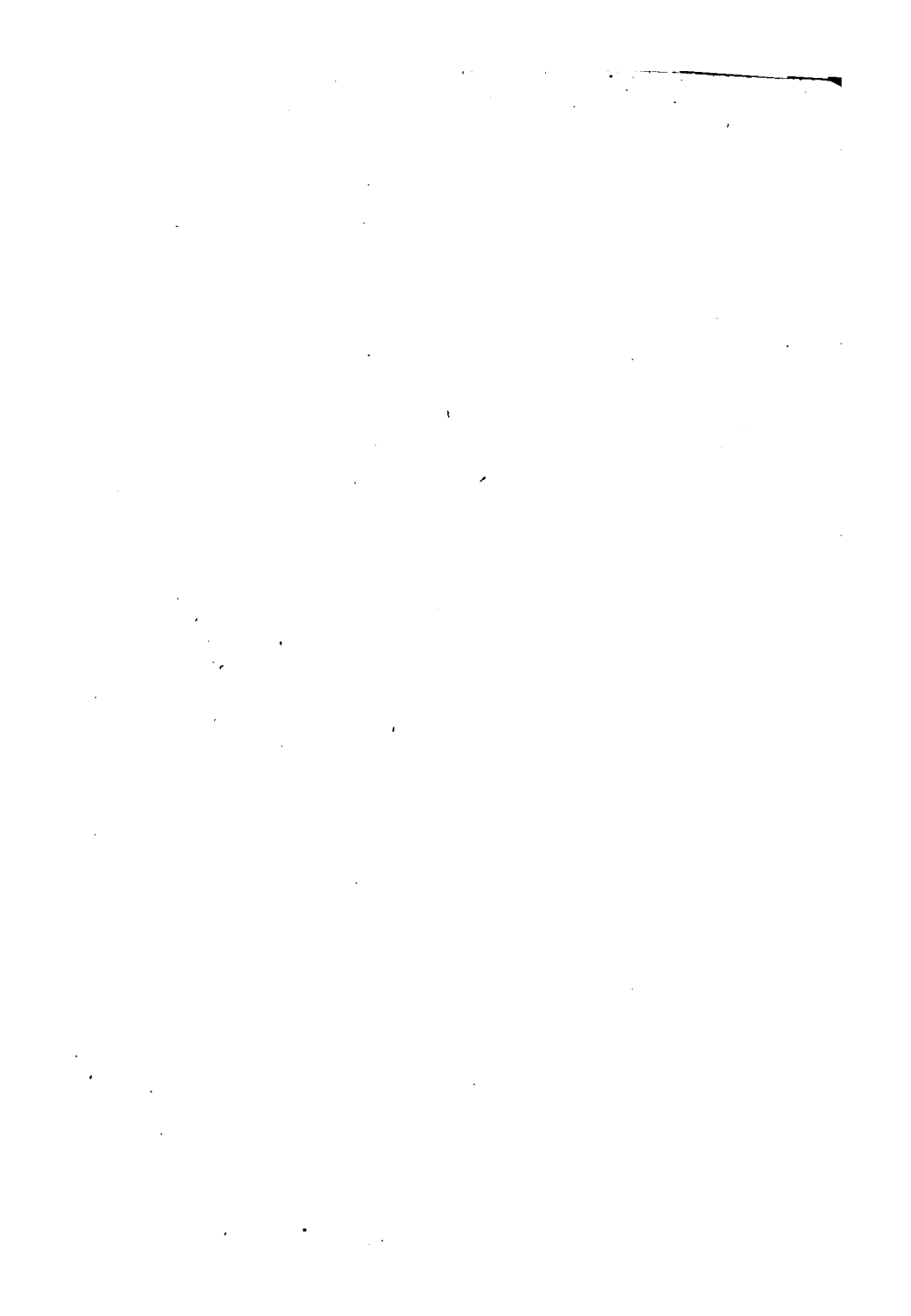
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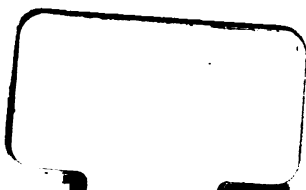
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